Training module

Recent Advances And Technique In Anaesthesia For Ss/Js In Raj. State Health System.
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FURTHER READINGS-
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2. BRITISH JOURNAL OF ANAESTHESIA
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# AIMS AND OBJECTIVES

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| 1   | ANAESTHESIA MACHINE, EQUIPMENT AND TECHNIQUES                           | • To introduce a new machine be reliable, easy to understand and operate, economical to run, require minimal servicing which can be carried out locally, and be versatile, so that the same machine can be used in all patients both as an anaesthetic machine in the operating room and as a ventilator in a recovery room.  
• To provide basic knowledge of recent vaporisers.  
• To upgrade the basic ideology of positioning of patient in theaters, to avoid various complications with conventional position.                                                                 |
| 2   | PHARMACOLOGY                                                           | • To provide informations of newer drugs usefull at 30 to 100 beded hospitals like, propofol, nitric oxides and levobupivacaine.  
• To know about the importance of acute oxygen treatment in critical situations in theater as well in recovery room.  
• To make familer with recent analgesics usefull in the management of pain for the patients suffering from variety of cancers at district level and provide palliative treatment, to reduce the burden over the tertery centers.                                                                 |
| 3   | ANAESTHESIA IN PATIENTS ASSOCIATED WITH COMORBID CONDITIONS             | TO STRENGTHEN THE KNOWLEDGE OF ANAESTHESIST AT DISTRICT AND LESS AFFULENT HOSPITALS TO PROVIDE ANAESTHESIA IN PATIENTS AT HIGH RISK VIZ. HEART DISEASE, HYPERTENSION, DIABETES MELLITUS, RENAL FAILURE AND LIVER DISEASES, THUS TO CURTAIL THE NUMBER OF REFERRAL TO THE TERTERY CENTERS. |
| 4   | ANAESTHESIA-SPECIAL COSIDERATION                                       | • To provide anaesthesia in mobile surgical camps.  
• To manage the patient of abdominal trauma very common in RTA at district levels including eye trauma.  
• To introduce certain newer aspects of GERIATRICS anaesthesia, since no. of patients requiring anaesthesia of age>70 years increasing, specially in village populations.                                                                 |
| 5   | REGIONAL ANAESTHESIA AND PAIN                                          | • To provide impitus for regional anaesthesia so that frequency of GA as well as its complications can be reduced.  
• To provide knowledge of nerve blocks  
• To provide analgesia in children postoperatively which is difficult and of its immense impotance.  
• TO START A NEW ERA OF PAINLESS DELIVERY.                                                                 |
EXPERIENCE WITH THE GLOSTAVENT ANAESTHETIC MACHINE

In many parts of the world, anaesthetists have to work in difficult or isolated situations where medical supplies are erratic and servicing facilities are poor or non-existent. Most modern anaesthetic machines, however, are not designed to be used in these conditions, as they require high levels of maintenance and servicing by trained engineers and are dependent on continuous supplies of compressed gases and electricity. Consequently, when conditions are unfavorable, they are liable to malfunction or even fail completely.

An anaesthetist working in such difficult conditions requires an anaesthetic machine which has been specifically designed to overcome these problems. It should therefore be reliable, easy to understand and operate, economical to run, require minimal servicing which can be carried out locally, and be versatile, so that the same machine can be used in all patients both as an anaesthetic machine in the operating room and as a ventilator in a recovery room or I.C.U. Most important of all, it must continue to function if either the electricity or oxygen supplies fail, situations that are all too common in parts of the developing world and that have been responsible for many tragedies.

The Glostavent anaesthetic machine has been designed to fulfill these requirements precisely.

In the development of the Glostavent, four separate components have been incorporated, each of which has, in its own right, already proved valuable in difficult environments. These are the draw-over anaesthesia system, the oxygen concentrator, the Manley Multivent ventilator, and the air compressor.

1. The Draw-Over Anaesthesia System
Atmospheric air is used as the carrier gas, which is drawn over a low resistance vaporiser, in this case, the Oxford Miniature Vaporiser (O.M.V.), either by the negative pressure created during inspiration in spontaneously breathing patients, or by the action of bellows when breathing is controlled. IT CAN THEREFORE BE COMPLETELY INDEPENDENT OF THE SUPPLY OF COMPRESSED GASES. Oxygen from a cylinder or a concentrator can be added upstream of the vaporiser, to increase the inspired oxygen concentration.

2. Oxygen Concentrator
This is an electrically powered device which produces a continuous supply of oxygen from atmospheric air, by first compressing the air and then directing it through canisters containing zeolite granules where the nitrogen is absorbed and the residual oxygen delivered to the patient. The zeolite granules are continually being re-activated and do not require changing.

3. Manley Multivent Ventilator
This is a pneumatically driven version of the Oxford inflating bellows. It can be driven either by compressed air or oxygen and only requires a volume of driving gas equal to 1/10 of the patient's minute volume. When oxygen is used for driving the ventilator, it is automatically collected and returned to the breathing circuit. In other words the same oxygen is used twice, first to drive the ventilator and then for the patient to breathe. The bellows of the Multivent can also be operated manually.

4. Air Compressor
This is an integral part of the oxygen concentrator, which has been modified to allow some of the compressed air generated by the concentrator to be diverted for use as driving gas for the ventilator, so that the concentrator provides both the driving gas for the ventilator and oxygen for the patient. In the design of the Glostavent, the four components are mounted on a single trolley, together with 2 reserve oxygen cylinders (figures 1 & 2). It was initially described under its original name of Oxyvent1 and subsequently as the Glostavent2. Although it has now been in regular use in several hospitals throughout the world for the past 6 years3 & 4, delivering anaesthesia safely to thousands of patients, few reports of its use have so far been published5, and a full description of its operation illustrating its many advantages is not available. Its potential as a safe, reliable and cost effective anaesthetic machine has been recognised by the Association of Anaesthetists of Great Britain &
Ireland, the World Federation of Societies of Anaesthesiology and the Department for International Development, all of whom have contributed to its development. However, anesthetists practicing in difficult situations or in isolation need to be confident that it will perform predictably and reliably in their own environments, so that it can be used safely when monitoring facilities are limited or totally absent.

**Paediatric Use**
The use of the draw over technique is not recommended in small children breathing spontaneously, because of the resistance of the circuit and the deadspace of the valves. For this reason, in children under 25kg, the Glostavent was converted for continuous flow use. This was achieved simply by occluding the open end of the reservoir tube with a bung, in order to allow the gas flow to build up sufficient pressure to pass through the vaporiser. A Mapleson E circuit was then attached to the common gas outlet, as with any standard continuous flow anaesthetic machine and oxygen administered at a flow rate of 4L/Min from either the cylinder of the concentrator. The O.M.V was shown to function equally satisfactorily for continuous flow and draw over anaesthesia, so that no change of vaporiser was required.

**Using the Glostavent**
An important feature of the Glostavent is its simplicity, enabling first time users to master it quickly and easily. The same circuit is used for both I.P.P.V. and spontaneous respiration. Conversion from one to the other simply involves turning the ventilator off and bypassing the bellows, no other action is necessary. A handle is attached to the bellows to facilitate manual ventilation when this is required.

Under normal circumstances, when electricity is available, it is more convenient as well as more economical, to conserve cylinders of oxygen and to use the concentrator to provide both the oxygen for the patient and, when I.P.P.V. is required, the compressed air to drive the ventilator. In this mode, a flow rate of oxygen of 2 L/Min delivered into the side arm of the reservoir tube raised the FiO2 to 50-55% in both spontaneously breathing and ventilated patients. This is satisfactory in most cases and can be recommended for routine use. Higher FiO2 values, in the region of 75% can be obtained by increasing the oxygen flow to a maximum of 5L/Min. If still higher oxygen concentrations are required, oxygen from the reserve cylinders can be added.

In the developing world oxygen cylinders are expensive to purchase and to transport and they should normally be kept in reserve, to be used only if an electricity failure renders the concentrator inoperable or to increase the FiO2 in an emergency.

When I.P.P.V. is used, the driving gas for the ventilator can either be oxygen from the cylinder (Group 3), or compressed air from the concentrator (Group 4). When the concentrator is in use, any interruption in the supply of electricity triggers an audible alarm. This alerts the anaesthetist that the concentrator has stopped. The reserve oxygen cylinders are then turned on and the anaesthetic can continue without interruption.

When cylinders are in use, conservation of supplies becomes extremely important. As has been clearly shown in groups 1 & 3, satisfactory FiO2's were achievable with minimal flows of supplementary oxygen and indeed during I.P.P.V. without the need for any additional oxygen whatsoever.

Further conservation is possible because of the unique design of the Manley Multivent ventilator. With most other gas driven ventilators, the volume of driving gas required is equal to the patient's minute volume. The Manley Multivent, however, was specifically designed for economy and the requirement for driving gas reduced to 1/10 of the minute volume. With the minute volume for example set at 4 litres per minute, the driving gas is utilised at a rate of 0.4 litres per minute and an E size oxygen cylinder containing 680 litres should not only be able to drive the ventilator, but also supply the average oxygen requirement for a period of 28 hours.

**Conclusion**
The Glostavent is much less expensive than the majority of continuous flow anaesthetic machines in current use and yet offers considerable advantages when used in difficult situations. These include, not only the low cost of the anaesthesia, but much more importantly, the ability to maintain the delivery of an anaesthetic safely when cylinders of oxygen, nitrous oxide and compressed air and supplies of sodalime may be scarce and the electricity supply unreliable.

Regardless of the conditions in which they work, the aim of anaesthetists all over the world is the same, that is to provide an anaesthetic service which is both effective and safe at all times. To achieve this, considerably greater demands are placed on anaesthetists in the developing world because of the lack of drugs, equipment and facilities than those in wealthy environments with greater resources. In the attempt to make anaesthetic machines ever more foolproof for use when conditions are ideal, the basic problems that still confront the majority of anaesthetists throughout the world are easily forgotten. Modern sophisticated machines, however expensive, cannot be considered good enough for use in the developing world if they cannot be relied on
when conditions become unfavorable. Only equipment which has been specifically designed to overcome their problems is adequate. The Glostavent can make a significant contribution towards meeting these requirements and is recommended for use in the developing world.

**Summary**
The Glostavent is an anaesthetic machine which has been designed to enable anaesthetists practicing in adverse conditions to overcome the difficulties they are likely to encounter. These include inadequate or non-existent monitoring and servicing facilities and frequent disruption in the supplies of oxygen, nitrous oxide, soda lime or electricity.

An examination of the records of patients who were anaesthetised using the Glostavent with full monitoring, demonstrates its predictability, reliability and economy over a wide range of clinical situations. Suggestions are made for its cost effective operation. It is recommended as an anaesthetic machine capable of providing a safe and reliable anaesthetic in adverse conditions.


**VAPORISERS**

**Early vaporisers**

Vaporizer rather than the word inhaler is used when we are talking of inhalational anaesthesia by continuous flow anaesthetic machines. Component parts of some early vaporizers, which have not been completely deciphered, are also on exhibit. They are Bernoy’s ether vaporizer (1), Ogeston’s chloroform vaporizer (2), Ohio no.8 ether vaporizer (3). Victor Goldman initially designed this in 1962 for administering halothane by intermittent flow machines (e.g. Walton V) for dental surgery. In India and the Armed Forces it found its maximum utility in continuous flow machines as a plenum vaporiser and as a draw over vaporizer in portable anaesthetic apparatus. Its use in closed circuit anaesthesia (VIC) in a spontaneously breathing patient has been described, but we don’t see it being used in this fashion. Its calibration at 30, 8 and 2 min-1 is therefore designed for its use in a particular circuit. Till date four versions of the vaporizers have appeared. The Mk IV has four notches and lever for change in concentration was made into a clicking device. In India prototypes of the same were manufactured by Khushwaqt and anaesthetics because of the exorbitant cost of there compensated vaporizers and introduction of halothane in the Indian market. They are still in use where sophisticated machines have not reached.

**Goldman type vaporizers**

The most common vaporizers incorporated in the anaesthetic machines available in India were the Boyle’s ether and trilene bottles. Many other vaporizers became popular before the tec series of vaporizers became standard. Though popular during the early 1970’s they are still being used in many places.

**Goldman vaporiser**

1 2 3

The one manufactured by Khushwaqt Industries. It is similar to Mk III of the original Goldman series. And the one manufactured by Anesthetics, except for size the design is quite different in that it has a screw on bottle, which tends to invariably chip at the screws. Some of the next generation vaporizers were the OMV, AE Vaporizer, EMO inhaler, and the PDV. All of these had low internal resistance and could therefore function’s draw over vaporizers. Apart from the primary agent these vaporizers incorporated scales for other agents. It is a temperature compensated vaporizer. Chloroforms probably as good as an anesthetic as halothane but it fell into disrepute and lost popularity altogether since accurate vaporizers for its delivery did not exist when it was introduced. This vaporizer probably marked there introduction of chloroform but never got a foothold.

**Penlon Draw over Vaporizer (PDV)**

Had been designed for methoxyflurane and has an additional scale for trilene. It was at one time popular in the Armed Forces forward surgical units. Its use declined with the withdrawal of methoxyflurane due to its fluoride toxicity. Earlier use of a single vaporizer that could be used for multiple agents was a desirable property of an idea vaporizer.

Manufactured by ‘Cyprane’ this was designed for halothane but has a dial for chloroform and trilene.

**Oxford miniature vaporizer (OMV)**

Was designed for halothane but has a scale for trilene. Two versions of OMV exist; OMV Fifty (Lt to Rt) for use in continuous flow machines and OMV Ten(Rt to Lt) for use in draw over anesthetic apparatus. Facility for temperature stabilization exist in this vaporizer.
POSITIONING ON THE OPERATING TABLE

Positioning patients is an important daily routine for anaesthetists to facilitate surgical access or a number of procedures. Different positions produce a range of physiological stresses. Particular care is needed for positioning anaesthetised patients to avoid passive movements that would not normally be tolerated. Nerve damage and pressure necrosis commonly result from poor positioning, the incidence is increased by hypotension and hypothermia.

Tourniquets can cause nerve damage if they are applied over a nerve trunk therefore the inflation pressure and the time of application should always be monitored. Diabetics, patients with arterial disease, the elderly and those with neurological deficits are also at particular risk.

Patients with rheumatoid arthritis may suffer from cervical spine instability at the atlanto-occipital level and it is important that their range of neck movement be assessed preoperatively. They should then be comfortably positioned prior to induction and this position maintained once anaesthetised. Sandbags may be employed.

Supine - “On the back”

The most common position. The arms should be carefully secured either next to the patient’s body, flexed across the chest or out on armboards. Acute flexion at the elbow may cause ulnar nerve damage due to trapping where it enters the cubital tunnel. The brachial plexus is a relatively fixed structure and therefore susceptible to traction injury. To avoid “stretch” on the plexus, pronate the forearms when the arms are extended by the patient’s sides. When both arms are abducted on boards, prevent over-abduction and hyperextension and keep the head facing forward. When one arm is abducted, the head should be turned towards that side, again to prevent traction on the brachial plexus (Figure 1). Legs should lie flat and uncrossed. A soft pad raising the heels from the table avoids pressure necrosis. Other sites susceptible to pressure damage are the sacrum and occiput and postoperative alopecia (hair loss) has been reported after long operations where hypotensive techniques have been employed. The patient’s eyelids should be carefully closed and taped to avoid corneal abrasion and dehydration. Direct pressure on the eye should be avoided as central retinal artery occlusion may occur. Ensure that no part of the breathing circuit, or other equipment, is pressing on the patient’s face.

Trendelenberg - “Head down”

Supine with head down tilt. This position is used in laparoscopic and varicose vein surgery.

Physiological effects of this position include:

- increased venous return
- Raised intracranial and intraocular pressure. Cerebral oedema and retinal detachment may occur if Trendelenberg is prolonged and steep. It is therefore important to avoid this position in a patient with potentially raised ICP.
- Lung compliance and functional residual capacity (FRC) are decreased with increased V/Q mismatch, especially in obese patients (IPPV may be preferable to SV)
- Increased intragastric pressure may result in reflux of gastric contents
- Venous stagnation with resulting cyanosis in the face and neck of plethoric patients

Reverse Trendelenberg - “Head up”

Supine with head up tilt. Reduced venous return in this position may lead to a fall in cardiac output and arterial pressure. As baroreceptor activity is reduced under anaesthesia a vasopressor may be needed. Blood pressure readings should be interpreted in the context of relative positions of the blood pressure cuff and the level of the brain above it. Functional residual capacity (FRC) is improved.
Lawn chair position (Figure 2)
Backache following anaesthesia is common and may occur from stresses on the interlumbar and lumbosacral ligaments, when the convexity of the lumbar spine is lost in the “lying to attention” position. The lawn chair position was developed to reduce this backstrain. The operating table is modified so that the patient lies slightly head up with hips and knees partially flexed. It is particularly useful for patients undergoing awake local anaesthetic procedures.

Prone - “Face down”
Used for spinal surgery, ligation of the short saphenous vein and some ankle operations. Intubation is normally required (although for short procedures a Laryngeal mask airway is sometimes used). A well-secured, armoured endotracheal tube is most suitable. Adequate eye protection and padding is vital because pressure on the eye can cause retinal artery occlusion and blindness.
A sufficient number of persons are required to turn the patient prone - the larger the patient the greater the number of assistants required. Usually 4 people will suffice: the anaesthetist to control the head, and 2-3 assistants to support the torso and arms, buttocks and legs respectively. The patient may be turned prone after transfer to the operating table or alternatively, turned in the process of the transfer. The head is positioned to one side or face down on a piece of hollow foam or headrest. Pressure should be limited to the forehead. Avoid any pressure on the eyes and ensure the endotracheal tube is secure. The arms are positioned fully adducted so they lie by the patient’s side or are abducted and flexed at the elbow so they lie alongside the head. Avoid undue pressure in the axillae as axillary nerve or brachial plexus neuropaxia may occur from overstretching (Figure 3).
Pressure points tend to be the head/face, anterior superior iliac spines, knees and feet which should all be well padded. Lung compliance is reduced due to decreased chest wall and diaphragmatic excursion. To aid compliance, a “Montreal” mattress (a rectangular mattress with a hole in its centre) may be used to prevent the abdominal contents forcing the diaphragm upwards. Alternatively, pillows should be placed under the iliac crests and chest, leaving the abdomen unhindered. This also prevents undue movement of the back and allows for efficient drainage from the epidural veins by reducing intrathoracic and intra-abdominal pressure. When using frames that support the anterior superior iliac spines, the lateral cutaneous nerve of the thigh may be compressed and stretched. The “Tarlov knee-chest position” (prone seated position) is a reliable position for lumbar surgery. The buttocks are supported on a “seat” and the table tilted upwards. This position rarely causes any damage other than an erythematous reaction on the skin of the knees.

Lithotomy - “Legs up”
Used for gynaecological and anal surgery. Both legs should be moved together to avoid strain on the pelvic ligaments and the knees should be positioned outside any metal supports. Avoid placing arms at the side as metal contact with the lithotomy pole may occur and trapping of digits in the lower section of the table is possible.
Potential problems include:
- common peroneal nerve damage due to compression between the head of the fibula and lithotomy pole if knee positioned inside the metal support
- saphenous nerve compression between lithotomy pole and medial tibial condyle
- autotransfusion from the leg vessels will increase preload. The effect on cardiac output will depend on the patient’s volume status
- vital capacity is decreased
• The risk of aspiration is increased therefore anaesthesia should never be induced in this position. Further, if reflux or vomiting occurs during induction, turning the patient will be delayed.

Lateral - "On the side"
Usually used for thoracotomies, renal, shoulder surgery and hip operations. The lateral position alters respiratory physiology: if breathing spontaneously, the dependent (lower) lung is efficiently perfused and ventilated. But with IPPV the dependent lung is better perfused and the non-dependent (upper) lung better ventilated, resulting in V:Q mismatch. Pressure points in this position are the dependent hip, shoulder and ankle and these should be padded where appropriate. The patient may be stabilised with chest and hip supports, or with a mattress which becomes rigid when air is evacuated from it. A pillow is placed between the legs, with the lower leg flexed at the knee and the upper leg in a neutral position. The upper arm may be allowed to hang freely above the head or placed in an arm support.

Lateral decubitus position for nephrectomy
The table is flexed in the centre in addition to the lateral position. The lateral decubitus position causes a V:Q mismatch as previously mentioned. This position can cause direct caval compression resulting in decreased venous return and hypotension. It is important to monitor blood pressure closely an arterial line may be useful. Pressure points are the dependent hip, shoulder and ankle. Once again, the patient is stabilised with chest and hip supports or with a mattress which becomes rigid when air is evacuated.

Sitting
Occasionally for posterior fossa neurosurgical procedures. It has a number of advantages over the prone position: better surgical access, more neck flexion, improved gravitational drainage of blood. Serious disadvantages include: postural hypotension, high risk of venous air embolism. This position has marked cardiovascular effects: cardiac output and arterial pressure may decrease dramatically due to pooling of blood in the lower extremities with resulting hypotension and reduced cerebral blood flow. Invasive monitoring (arterial/central lines) is required. In order to minimise the effects of changing from supine to a sitting position, a number of measures can be taken. These include fluid loading, compression stockings or G-suits and/or the use of vasopressors. The patient should be raised slowly with elevation of the legs above the horizontal once in the sitting position to aid venous return. The head may be supported in a horseshoe headrest that allows pressure to be applied to the head and neck without movement. Alternatively a skull clamp may be used which minimizes pressure-related complications involving the face. Its insertion is stimulating but may be attenuated by using local anaesthetic, a small bolus of propofol or a short acting opioid. Flexion of the head on the neck aids surgical access, but raises ICP and may cause swelling of the face and tongue because venous return is decreased. These patients are at marked risk of air embolism and should be monitored using ETCO₂, Doppler, transoesophageal ECHO or oesophageal stethoscope. The risk is reduced by IPPV and by maintaining mean arterial pressure. Spontaneous ventilation is permissible (indicates that respiratory centre is intact), but is seldom used now. Armchair position for shoulder surgery
Occasionally an armchair position is adopted for surgical access. This position has cardiovascular effects similar to the sitting position namely, hypotension due to pooling of blood in the lower limbs. These effects can be reduced by elevating the patient to a head up position slowly, using vasopressors and fluids as required, and elevation of the patient’s legs above the horizontal. These patients are also at risk of air embolism.

<table>
<thead>
<tr>
<th>Nerve</th>
<th>Site of Potential Damage</th>
<th>Result of Damage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Supraorbital numbness of</td>
<td>Compression from a tight facemask</td>
<td>Photophobia, pain in the eye, the forehead</td>
</tr>
<tr>
<td>Facial</td>
<td>Lies superficially and may be damaged at the ramus of the mandible</td>
<td>Paralysis of the face and orbicularia (buccal branch)</td>
</tr>
<tr>
<td>Axillary</td>
<td>Prone to stretching when shoulders are lateral aspect of upper head (prone position)</td>
<td>Decreased abduction of arm, reduced skin extended and arms placed above the sensation over arm.</td>
</tr>
<tr>
<td>Radial</td>
<td>At risk of external pressure in axilla if arm hangs over the edge of table (posterior cord)</td>
<td>Wrist drop</td>
</tr>
</tbody>
</table>
Median: Very uncommon injury. At risk of direct needle trauma in artecubital fossa. Inability to oppose thumb and little finger.

Ulnar: May be compressed by edge of operating mattress where it lies superficially in groove behind medial epicondyle of humerus. Internal compression between two heads of flexor carpi ulnaris. Full flexion at elbow causes compression where the nerve enters the cubital tunnel. Hand weakness, tingling and pain.

Sciatic: Main source of damage direct trauma from misplaced i/m injections pneumatic tourniquet. Paralysis of all muscles and sensory loss below knee.

Femoral: Susceptible to damage where it passes extension, loss of sensation over anterior thigh and anteromeaspect of calf beneath inguinal ligament. Excessive leg flexion in lithomy may cause entrapment. Lateral cutaneous nerve of the thigh common peroneal. Loss of hip and knee.

Pudendal: Compression against perineal post used in hip surgery. Loss of perineal sensation, faecal incontinence.

Saphenous: Compressed between medial tibial condyle and lithotomy pole (leg lateral to pole).
NITRIC OXIDE AND PROPOFOL

Abstract
Propofol, an intravenous anesthetic, is similar in chemical structure to the active nucleus of antioxidant substances such as alpha-tocopherol (vitamin E), butylhydroxytoluene and acetylsalicylic acid (aspirin). Recent studies have demonstrated that some effects of propofol may lie in its antioxidant properties and the likely involvement of nitric oxide. This review article focuses on the relationship between nitric oxide and propofol. There is an implication that nitric oxide is responsible for the hemodynamic responses of propofol. The antioxidant effect of propofol may also extend its anesthetic application.

Introduction
Propofol is a rapidly acting intravenous anesthetic agent that has gained widespread acceptance for anesthesia and sedation. The rapid and complete recovery profile associated with propofol offers advantages over other injectable anesthetic agents (1,2). Administration of propofol produces pronounced hemodynamic responses, particularly the decrease in arterial blood pressure (3-11). Published reports have not agreed as to the mechanism of propofol-mediated hypotension, but some investigators have attributed significant decrease in systemic vascular resistance to the blood vessel-dilation property of propofol to (12,13) with the likely involvement of endothelium-derived relaxing factor (nitric oxide) (14). Most importantly, propofol has been found to bear antioxidant capacity due to its structural similarity to tocopherol (15-43), which is strongly related to free radicals. At this point propofol is not only an anesthetic agent but also an antioxidant drug. In this review, we have focused on published literature relating to free radical nitric oxide and its oxidative reaction with propofol.

Formation, decomposition and reactivity of nitric oxide
Nitric oxide (NO) is an inorganic gas synthesized by the enzyme nitric oxide synthase (NOS) in which amino acid L-arginine is oxidized to NO and equal amount of L-citrulline (44-46). Three NOS isoforms have been identified: neuronal NOS (nNOS), endothelial NOS (eNOS), and iNOS (47). The former two isozymes are constitutively expressed (constitutive NOS) and have physiologic roles; the last is usually present only after the induction by inflammatory stimuli.

The genes encoding eNOS, nNOS and iNOS have been cloned and sequenced (47-49). These isoenzymes are distinct and located on different chromosomes (7, 12 and 17, respectively). They are structurally related to the cytochrome p-450 supergene family and consist of a single polypeptide chain containing L-arginine, heme and calmodulin binding sites as well as a complete NADPH diaphorase. Required co-factors include oxygen, NADPH, calcium (constitutive NOS) and tetrahydrobiopterin (50, 51).

Neuronal NOS (nNOS) is mainly distributed in the nervous system. NO from nNOS functions as a neurotransmitter (52-58) in long-term potentiation (59), gonadotropin secretion (60-64), sexual behavior (65-69), regulates emotional behaviors (70, 71) and autonomic outflow to the cardiovascular system (72,73). nNOS is also present in the kidney (74), skeletal muscle (75-77) and myocardium (78), as well as pancreatic islets (79, 80).

Endothelial NOS is predominately present in the endothelium of blood vessels. NO released from endothelium is found as endothelium-derived relaxing factor, or EDRF, which modulates vascular tone and accommodates change in blood flow (81-84). Endothelial NOS is also present in some immune cells (85, 86). Accordingly, eNOS "knockout" mice are routinely hypertensive (87-93).

Inducible NOS is expressed in tissues of the immune system (macrophages, leukocytes and other phagocytic cells) on stimulation with cytokines and/or endotoxin (94-96), vascular smooth muscle (94, 97), endothelium (97, 98), kidney (mesangium, tubules) (99,100) and other sites (pancreas, liver, enterocytes, airway, pneumocytes) (101-113). NO from iNOS is present in a large amount. It exerts antimicrobial, cytotoxic effects and immunoregulation (cytokine production, apoptosis and signalling) in the immune system. Therefore, inducible NOS knockout mice exhibit loss of immune function and minor hypotension (113-121).

NO functions by diffusion to kill bacteria and other microbial pathogens (122-124) or possibly acts at the enzyme guanylyl cyclase levels and augmenting cyclic guanosine monophosphate (cGMP) production (125-129). Compared with neurotransmitter receptors or related adenylyl and guanylyl cyclases (130,131), the NO receptor enzyme appears rather unremarkable. It is composed of two different subunits, but only two isoforms have been shown to exist at the protein level: the 11 isoform, which is expressed widely, and the 21 isoform present in human placenta (132-134). Several receptor systems, including N-methyl-D-aspartate (NMDA) (135, 136), muscarinic (137, 138), and gamma-aminobutyric acid (GABA) receptors (139, 140) and A2-adrenoceptors (141), have been shown to mediate their action via the NO-cGMP pathway.
NO is a labile species with a half-life of only a few seconds in biological systems. It degrades rapidly to NO2- (nitrite). Nitrite is unstable and it is further converted to the end product NO3- (nitrate). Putative intermediate metabolites include an array of low and high molecular weight thiol--nitrosoglutathione, nitrosalbumin, S-nitrosohaemoglobin (142). This is not only a mechanism for scavenging NO but also serves to transport NO and is the molecular basis for biological effects in its own right. In the presence of O2, NO reacts with O2- to form ONOO- (peroxynitrite) and other NO radicals as well. Overproduction of NO can lead to cytotoxicity. NO rapidly oxidizes sulphydryl groups and thiethers in peptide, proteins and lipids (143). In addition, NO nitrates and hydroxylates aromatic compounds, including guanosine (DNA damage) (144), benzene (145, 146), tyrosine (147), tryptophan (148), 4-hydroxyphenylacetic acid (149), and tocopherol (150). These deleterious effects of peroxynitrite may disturb cell-signalling processes (Fig. 1). Structural formulae of propofol, alpha tocopherol, butylhydroxytoluene and acetylsalicylic acid. There is a hydroxyl substituent and a benzene group in all four compounds.

Propofol and its clinical relevance with NO

Propofol (2,6-diisopropylphenol) is an intravenous anesthetic that is widely used for both induction and maintenance of general anesthesia. The pharmacokinetics of propofol is best described by a three-compartment model: the central compartment, the shallow peripheral compartment and the deep peripheral compartment. Of the greatest importance is the rapid clearance of propofol (rapid and complete recovery), which is approximately ten times faster than that of thiopental. This made propofol the best controllable intravenous hypnotic from a pharmacokinetic point of view (1,2, 151). Its clinical uses include ambulatory anesthesia, monitored anesthesia care, neuroanesthesia, cardiac anesthesia, pediatric anesthesia and sedation in the intensive care unit (2).

Use of propofol for induction of anesthesia causes decrease in arterial pressure and systemic vascular resistance. Systolic arterial pressure (SAP) is decreased after the start of induction; diastolic pressure (DAP) is decreased at 60 s after start of induction and further decreases are seen until 210 s after induction (6). The precise mechanism(s) of propofol-induced hypotension is not known. Many studies have attributed the hypotensive responses to decreases in peripheral resistance. This can be prevented by effective volume loading (152), but cannot be attenuated by administration of a fluid preload (10). Induction of anesthesia with an opioid-benzodiazepine combination followed by a maintenance infusion of propofol, supplemented with an inhalational agent or opioid analgesic or both, appears to control blood pressure as well (2). Some studies suggested that propofol-mediated hypotension is due in part to an inhibition of the sympathetic nervous system (153) and to an impairment of baroreflex mechanism (154). A reduction in plasma norepinephrine concentrations after propofol has been demonstrated also (155). Recently, a possible involvement of endothelium-derived relaxing factor or nitric oxide was proposed in the rapid onset of vasodilatation produced by propofol (14). It was reported that propofol stimulated nitric oxide release from cultured porcine aortic endothelia cells and an inhibitor of NO blocked the effects of propofol (156). In a different study, propofol showed a contractile effect in isolated aortas from spontaneously hypertensive rats in the present of a nitric oxide inhibitor (157). However, another study examined the effects of propofol on rat aortic and pulmonary artery rings and demonstrated a marked relaxation, which was endothelium-independent (158). In addition, the same effect was observed in isolated mesenteric arteries from humans. Further studies on the mechanism responsible for the reduction in systemic vascular resistance and hypotension of propofol are needed.

Antioxidant activity of propofol

Free radicals are believed to contribute the tissue injuries associated with many pathological processes such as ischemia, tissue anoxia, inflammatory process, infection, carcinogenesis, neurodegenerative disorder and diabetes (160-162). In such diseases, antioxidants can protect tissues by inhibiting lipid peroxide formation or increasing the activity of the glutathione antioxidant system, among other mechanisms.

Propofol has a structure (2,6-diisopropylphenol) similar to that of known antioxidants (Fig. 2), such as tocopherol (vitamin E), acetylsalicylic acid and butylhydroxytoluene.

The ability of propofol to inhibit the formation of lipid peroxides has been found in several media in which free radicals are produced, e.g., liver and brain microsomes in the rat (16), liver mitochondria in the rat (18, 23), and chemical media enriched in arachidonic acid or linoleic acid (Using normal rat tissues (36) and in an vitro model of cerebral anoxia in the rat, it was found that the antioxidant effect of propofol is manifested not only as an inhibition of lipid peroxidation, but also as a decrease in tissue consumption of glutathione.

Studies in animals show that propofol, indeed, reduces the formation of lipid peroxides. In humans, there was no effect on plasma lipid peroxide levels in patients given propofol. However, others showed an increase in plasma antioxidant capacity in patients anesthetized with propofol. The highest levels of peroxides occur in cell membranes, rather than in plasma, and the antioxidant glutathione pathway is an important intracellular antioxidant system. In a group of surgical patients who were given propofol anesthesia, propofol showed antioxidant effects as evidenced by...
the inhibition of lipid peroxidase production in the platelet membrane and changes in the glutathione antioxidant system). In other experiments, propofol enhanced red blood cell antioxidant capacity in swine and humans.

Propofol, like other phenol-based antioxidant compounds, also acts directly as a free radical scavenger. Studies on the ameliorating effect of propofol in inhibiting radical production revealed that it preferentially scavenges organoradical species. In aqueous suspension it is more efficient than butylated hydroxytoluene (BHT) as a free radical scavenger of riboflavin radicals and in blocking formation of malondialdehyde degradation products generated from lipid hydroperoxides of arachidonic acid (20). In additional experiments it was found, using electron spin resonance (ESR), that propofol reacted with oxygen free radicals or peroxynitrite to form phenoxyl radical). Moreover, it was demonstrated employing mass spectrometry, that propofol could react with NO to generate nitro-propofol in vitro (forming phenoxyl radical)). Thus, propofol is a peroxynitrite scavenger. Because of these reactions, propofol has neuroprotective properties against injuries caused by ischemia/reoxygenation. Also, propofol prevents and reverses the inhibition of excitatory amino acid uptake in astrocytes exposed to tert-butyl hydroperoxide. The ability of propofol to defend against peroxide-induced inhibition of glutamate clearance may prevent the pathologic increase in extracellular glutamate at synapses, and thus delay or prevent the onset of excitotoxic neuronal death).

Furthermore, propofol had a protective effect in neurons against acute mechanical injury. A water-soluble prodrug of propofol protects from neuronal cell death from oxidative injury caused by glutamate (43). This is consistent with the clinical observation that use of propofol is associated with significant cerebral protection. The same protection was obtained in heart reperfusion injury. Isolated perfused Wistar rat hearts were subjected to either warm global ischaemia (Langendorff) or cold St. Thomas' cardioplegia (working heart mode) in the presence or absence of propofol. It was found that with the presence of propofol the heart injuries were significantly less, probably as a result of diminished oxidative stress (172). In isolated, working rat hearts submitted to ischemia, followed by reperfusion, it was observed that propofol attenuated mechanical dysfunction, metabolic derangement, and lipid peroxidation during reperfusion (24, 173). Additional experiments demonstrated that in vivo, propofol ameliorated dysfunction of the myocardium but not of the coronary endothelium resulting from brief ischaemia and reperfusion. The protection may be related, at least in part, to its ability to reduce lipid peroxidation (174).

However, the antioxidant properties of propofol are different depending on the formulation of propofol. Propofol inhibited the chemiluminescence (CL, a measure of oxidative stress) produced by stimulated polymorphonuclear (PMN) leukocytes in a dose dependent manner (until 5 x 10^-5 M, a clinically relevant concentration), while Diprivan (the commercial form of propofol) and intralipid (IL, vehicle solution of PPF in Diprivan, composition: 1.2% egg phosphatide, 2.25% glycerol) were not dose-dependent inhibitors. The CL produced by endothelial cells was dose-dependently inhibited by Diprivan and PPF, and weakly by IL (not dose-dependent). In cell free systems, dose-dependent inhibitions were obtained for the three products with a lower effect for IL. Diprivan efficaciously protected endothelial cells submitted to an oxidant stress, while IL was ineffective. By HPLC, it was demonstrated that PPF was not incorporated into the cells. The drug thus acted by scavenging the active oxygen species released into the extracellular medium. IL acted in the same manner, but was a less powerful antioxidant (38).

In conclusion, there is an implication that propofol enhances NO production in vascular system and that NO is probably responsible for the hypertension. The unique antioxidant ability and free radical scavenger of propofol may lead to further broaden its current clinical application in the future.
**LEVOBUPIVACAINE**

**A long acting local anaesthetic, with less cardiac and neurotoxicity**

**Introduction**

The property of isomerism occurs when two or more compounds have the same molecular composition, but a different structure which often results in different properties. There are two types of isomerism - structural and stereoisomerism.

Structural isomerism means that the compounds have the same molecular formula, but a different chemical structure. This may result in the compounds having similar actions like the anaesthetic volatile agents isoflurane and enflurane or different actions like promazine and promethazine. Stereoisomerism describes those compounds which have the same molecular formula and chemical structure, but the atoms are orientated in a different direction. There are two isomers, each a mirror image of the other, called enantiomers. They are also called optical isomers because they rotate the plane of polarised light either to the right referred to as +, dextro, d or D isomer, or to the left referred to as -, laevo (levo), l or L isomer. More recently this classification has been replaced by the R/S- notation, which describes the arrangement of the molecules around the chiral centre (R is for rectus the Latin for right, and S for sinister, left). The R enantiomer rotates light to the right and the S enantiomer to the left. As with other isomers, they can have different properties.

The molecule of bupivacaine, a long acting local anaesthetic, has an asymmetric carbon atom. For this reason, with this asymmetric carbon as a chiral centre, bupivacaine exhibits this phenomenon. In the commercial presentation of this local anaesthetic there is a 50:50 proportion: levobupivacaine, L (-) isomer, and dextrobupivacaine D (+) isomer. This preparation which contains both enantiomers is called a racemic mixture. The preparation of levobupivacaine contains only the levorotatory isomer present in the commercial preparations of bupivacaine.

Interest in levobupivacaine arose after several cases of severe cardiotoxicity (including death) were reported where it was shown that the D isomer of bupivacaine had a higher potential for toxicity. Consequently, it was thought that if it was possible to use only the levorotatory isomer, levobupivacaine, the risk for cardiac and neurotoxicity could be less than with the racemic bupivacaine but with similar clinical effects.

**Chemistry**

Here we will expose some general concepts about all local anaesthetics, with a special reference to levobupivacaine. Local anaesthetic molecules all have three characteristic portions:

- **A benzene ring - aromatic head**
- **An intermediate chain**
- **An amino group**

The benzene ring is very soluble in lipids.

The intermediate portion, a bridge between the other two, can have one of two types of chemical structures: Ester (COO⁻), or Amide (CONH⁻) (figure 2). Therefore, chemically, there are two large groups of local anaesthetics, depending on this intermediate portion of the molecule: Ester type and Amide type local anesthetics. Procaine is the prototype of the first group (figure 3), and lignocaine is the prototype of the second one (figure 4). The first group more commonly cause allergic reactions and have a short length of action as they are rapidly metabolized by cholinesterase. In contrast the second group, amides, rarely cause allergic reactions but are more likely to cause toxic reactions if the dose is exceeded. Levobupivacaine is an amide, which like the other amides, is a weak base. Depending on the pH, the amino group can adopt the tertiary or the quaternary form. The drug is in dynamic balance between the tertiary form, a free base, and the quaternary form, which has a positive charge, making it very water-soluble.

The pKa of levobupivacaine is 8.1, similar to the pKa of the racemic bupivacaine. (pKa is the pH at which 50 % of the molecules are free base and 50 % of the molecules have a positive charge - ionised). If bicarbonate is added to levobupivacaine, the pH is increased leading to a rise in the percentage of free base molecules. Those molecules cross more easily through the axon membrane and the pharmacological action begins more quickly.

In contrast, if the pH is low (acid), as happens when there is a local infection, there will be less free base molecules to cross the axon membrane resulting in smaller action over the axon.

Protein binding of levobupivacaine is more than 97%, mainly to alpha1-glycoprotein, rather than to albumin. This union to proteins is somewhat higher than the union of racemic bupivacaine to proteins (95%). This means that less than 3% is free in plasma. It is the free levobupivacaine, a small fraction of the total concentration that can have an action on other tissues, causing the unwanted side-effects, and producing the toxic manifestations. In hypo-proteinaemic, undernourished patients, patients with the nephrotic syndrome and in the newborn there is less protein for binding, causing higher levels of free drug, resulting in toxic effects being seen at lower doses.
Bupivacaine has stereoisometric properties as explained earlier. Commercial production of levobupivacaine for clinical use was started because it was observed experimentally that the D isomer had a lower threshold for causing tachycardia and dysrhythmias, which include, AV block, QRS widening and ventricular tachycardia and fibrillation than either the L isomer or the racemic preparation.

The levo isomer was used in rats and its effect was compared with the dextro isomer. It was found that with doses of 2mg/kg, all the animals of the dextro group developed apnoea, bradycardia, hypotension and finally died. In contrast, no animal in the levobupivacaine group had apnoea and only 30% had a slight bradycardia. In sheep experiments in which racemic bupivacaine was administered in toxic quantities, it was found that the concentration of the dextro isomer was higher in the myocardium and brain than the concentration of the levo isomer. This work together with other similar studies, led the investigators to conclude that levobupivacaine was less toxic than the racemic bupivacaine, but with similar clinical activity. Additionally, electrophysiological studies have been made which demonstrate that blockade of the inactive sodium channels is stereoselective, with the D isomer being more potent and faster than the L isomer. As this includes the cardiac fibres, it explains the higher cardiotoxicity associated with the D isomer.

Some of the first clinical studies in humans in Brazil, compared the effects of the racemic preparation and the levo isomer of bupivacaine when given peridurally. No significant difference in onset time, quality of anaesthesia and level of blockade has been found.

**Pharmacokinetics**

There are difficulties in carrying out pharmacokinetic studies with bupivacaine. Classic pharmacokinetic studies are usually performed using an intravenous application of the drug. These studies are more accurate because there are fewer possible causes of error, than when other access routes are used, such as intramuscular or subcutaneous infiltration. With both these routes of administration, the rate of absorption is an important but unknown factor affecting the rate of absorption between patients with different pathologies. In addition intravenous administration of bupivacaine or levobupivacaine, for pharmacokinetic studies has limitations, because of the risk of fatal toxicity. Additionally, in clinical practice this drug is not used intravenously.

Practical clinical studies have been carried out giving the drug for epidural and regional blocks. Placental transfer of levobupivacaine is similar to that of bupivacaine resulting in lower risk to the fetus. Like racemic bupivacaine, levobupivacaine is metabolised in the liver, primarily by the cytochrome P450, specially the CYP1A2 and CYP3A4 isoforms. Clearance is reduced when the hepatic function is damaged.

**Pharmacodynamics**

The mechanism of action of levobupivacaine is exactly the same as that of racemic bupivacaine and that of all the local anesthetic drugs in clinical use today. When the minimum local analgesic concentration (MLAC) close to the membranes of the axons is reached, the molecules block the sodium channels, in the resting position. In this way, the transmission of the nerve impulses stops.

This action is produced with an onset very similar to that of racemic bupivacaine. The duration of action is also similar to that of the racemic substance. Recent research work has been directed at the toxicity associated with the levo isomer, and how it compares with the racemic preparation. Differences were found between the two isomers. The concentration necessary to produce cardiac and neurotoxicity is higher for levobupivacaine than for racemic bupivacaine. The safety margin is estimated at 1.3 which means that toxic effects are not seen until the concentration rises by 30%.

**Toxicity**

Volunteers have been given bupivacaine or levobupivacaine intravenously at a rate of 10mg/min, until the appearance of early symptoms of central nervous system toxicity. These appeared at a lower dose (Mean 47.1mg) with bupivacaine than with levobupivacaine (56.1mg). Similarly there was a greater reduction in the myocardial ejection fraction and systolic and acceleration index with racemic bupivacaine when compared to levobupivacaine.

When 40mg of either levobupivacaine or racemic bupivacaine were administered over a 10min period, the EEG was significantly slower after racemic bupivacaine. Thus at similar doses, electrical activity is more affected by racemic bupivacaine. Levobupivacaine appears to cause less myocardial depression than both bupivacaine and ropivacaine, despite being in higher concentrations.

**Clinical Applications**

Levobupivacaine has been introduced into clinical practice within the last few years. It has been used at all sites: epidural, subarachnoid, different levels of brachial plexus block - interscalene, supra and infraclavicular, intercostals and peripheral nerve blockade, peribulbar and retrobulbar blockade, local infiltration, obstetric analgesia, postoperative pain management, acute and chronic pain management. The doses used are very similar to those of bupivacaine. As a result of its lower cardiac and neurotoxicity compared to racemic bupivacaine, anaesthetists feel safer working with levobupivacaine, than with bupivacaine.
Nevertheless, we must always remember that it is still a potentially toxic local anaesthetic. The initial licensing authority recommended a maximum single dose of 2mg/kg, and 400mg (5.7mg/kg) in 24h. Since then, some studies have shown that higher doses are safe, but further work is required. Special caution is recommended for hypoproteinemic patients, including adults with nephrotic syndrome, severe hepatic disease and the newborn. In Colombia, we have been using this new local anaesthetic for a year, with excellent results.[3] We only have the 0.75% formulation and use almost the same dose as when using bupivacaine. We have had no reports of toxic reactions. During the Colombian Congress of Anesthesiology (2001), a paper was presented, comparing levobupivacaine with ropivacaine in epidural anaesthesia. The two drugs were comparable, with a very good quality of epidural anaesthesia. However there were three cases of bradycardia in the levobupivacaine group which were treated successfully with atropine. The duration of the motor blockade in the postoperative period was less than after racemic bupivacaine.

Adverse Effects
These are the same as caused by racemic bupivacaine and any other local anaesthetic. They include hypotension, bradycardia, nausea, vomiting, pruritus, headache, tinnitus, dizziness, constipation, vomiting and convulsions. There have been reports of cases where the drug has been given in higher doses than that recommended, with no apparent toxicity. In one case, a single dose of levobupivacaine of 250mg for a brachial plexus block, far exceeding the maximum recommended dose (150mg), without toxicity symptoms, although further data will be needed before the safety of this level of dosage is confirmed.

There is a report where approximately 1.7mg/kg racemic bupivacaine was injected probably by an accidental intravenous injection during an attempted supraclavicular brachial plexus block. The patient lost consciousness, developed a tachycardia, hypertension and generalized twitching, was managed with oxygen and propofol, with a successful recovery after a few minutes with no sequelaes.[4] The authors stressed the risks associated with administration of high doses of bupivacaine, even in experienced hands and underline the need for possibly safer agents such as levobupivacaine.

Conclusion
Levobupivacaine is a relatively new long acting local anaesthetic, with a pharmacological activity very similar to that of racemic bupivacaine. The first studies in humans confirm the animal studies and the in vitro studies, which showed that this new molecule is less cardiotoxic and less neurotoxic than the racemic bupivacaine. Levobupivacaine can be used for all indications in which the anaesthetist needs a long acting local anaesthetic. The reduced toxicity of this new local anaesthetic is an advantage for the patient. The cost in Colombia is 40% higher, than racemic bupivacaine.
A NEW MANDATE FOR THE ANESTHESIOLOGIST-CANCER PAIN SPECIALIST: EXPERTISE IN PRESCRIBING ANALGESICS

Introduction
The anesthesiologist with an interest in the management of cancer pain may make important contributions to the well being of the cancer patient with pain, as well as to the effectiveness of teams organized to deliver pain and symptom control. Traditionally, the role of the anesthesiologist has been somewhat narrowly conceived of as that of a skilled technician providing invasive procedures when warranted, a "job description" related in large part to the skillful adaption of regional anesthetic techniques to the management of chronic symptoms. The practice of cancer pain management has evolved considerably over the last decade, mandating careful scrutiny of the anesthesiologist's role in contemporary cancer pain management. This article outlines a rationale for an expanded role for the anesthesiologist-pain specialist and reviews the basic aspects of the pharmacologic control of cancer pain.

Basis for Expanded Role of the Anesthesiologist
The rationale for adopting a broader view of the anesthesiologist's contribution to cancer pain control relate to several fundamental concepts that have come to serve as foundations for the contemporary practice of cancer pain medicine, hospice care and palliative care. These factors can be conceived of as including: (1) the acquisition of new pharmacologic knowledge and consequently, a greater emphasis on pharmacotherapy, (2) a reappraisal of the roles of invasive versus noninvasive interventions that views them as complementary rather than mutually exclusive; (3) an emphasis on quality of life as the end point of a broad based therapeutic armamentarium, the goals of which include the management not just of pain, but also of other symptoms.

A New View of Opioid Pharmacotherapy
Foremost among these factors is a reappraisal of the pharmacology of chronically administered opioids, and a growing understanding of the significant differences in pharmacology that exist with chronic as opposed to acute administration. Most of the knowledge gleaned about opioid pharmacology, until recently, was derived from single or limited dose studies conducted in the presence of either experimentally induced or acute pain. In a construct that recognizes chronic and acute pain as distinct disorders, there is limited justification for applying knowledge gained from one setting to the other uncritically. The inadequate scientific basis for prescribing practices is linked to and compounded by firmly held beliefs regarding the dangers of opioid therapy. Such beliefs are now widely understood to be based more on cultural biased than medical considerations. Recognition that the uncritical acceptance of these biases has historically impeded legitimate scientific inquiry and the dissemination and implementation of knowledge already on hand has engendered an almost unparalleled scientific activism to dispel these myths. Guidelines released by the World Health Organization, American Pain Society, American Society of Clinical Oncology, Oncology Nursing Society the American Society of Anesthesiologists and U.S. government Agency for Health Care Policy Research, stress the importance of opioid therapy and articulate the need to overcome exaggerated concerns about its risks.

Through the Lookingglass: "Oral Opioids Are Ineffective"
In prior eras oral morphine was deemed ineffective, with the first edition of the A.M.A. Drug Evaluation Manual (1971) referring to orally administered morphine simply as "not recommended." Biases against the use of oral opioids can be traced to an incomplete understanding of their clinical properties, especially the concepts of parenteral:oral bioavailability, lack of ceiling doses and a propensity for the rapid development of tolerance to most adverse effects. An illustrative study was conducted by Beecher and colleagues. They compared the effectiveness of 10 mg oral morphine, aspirin and placebo in postoperative patients, and demonstrated that morphine was no more effective than placebo and was less effective than aspirin. Having administered oral morphine in doses expected to be therapeutic, they concluded that oral morphine was ineffective as an analgesic. Only in light of later studies demonstrating the limits of oral morphine's bioavailability, does it become apparent that Beecher's study utilized sub-therapeutic doses of oral morphine, and as such simply provided preliminary indirect evidence of a parenteral:oral analgesic equivalency of greater than 1:1. Other contemporary research has demonstrated a lack of ceiling effect to opioid-mediated analgesia and a high incidence of the rapid development of tolerance to adverse effects such as nausea and sedation.

The Contemporary View: Oral Opioids as Treatment of First Choice
Oral morphine and its congeners were not widely used until the experience gleaned from the British hospice movement in the 1960's exerted its influence during the 1970's and 1980's. In large part as a result of the World Health Organization's identification of cancer pain as a major global health problem, well controlled, cross culturally validated trials of oral pharmacotherapy have since been performed that demonstrate efficacy in 70% or more of patients with cancer pain. Such efficacy combined with the favorable safety profile of oral opioids
Advances in microprocessor technology and the maturation of organized home care, together with the more facile access to medical care, have resulted in an intermediate role for subcutaneous and intravenous opioid infusions (with or without patient controlled analgesia), especially for patients with alimentary dysfunction. These same trends, together with the introduction of intraspinal opioid therapy have made an entire set of new modalities available for both ambulatory and bedbound patients. While neural blockade remains an important option for selected patients, parenteral and intraspinal opioid therapy have the advantages of titratability, reversibility and efficacy for generalized and multifocal pain. The latter feature is particularly important in virtue of the fact that most cancer patients experience more than one distinct pain problem.

Enhanced interest and knowledge about cancer pain and the more liberal use of opioids has had another important effect on the indications for neural blockade. In the context of an historical view that considered the use of opioids as being prima facie undesirable, there was a tendency to correlate the outcome of nerve blocks with whether opioid use could subsequently be discontinued or dramatically reduced. With the current emphasis on quality of life, independent of opioid doses per se,18,28 nerve blocks are more readily viewed as occupying a role that complements rather than replaces that of opioids. Reductions in opioid use are often still sought as a means to reduce drug side effects and as indirect evidence that the correct procedure has been selected and properly executed. Nevertheless, efficacy is not generally judged directly in light of changes in dose requirements, but instead on clinical reports of pain and toxicity.

Juxtaposing opioid therapy and neural blockade as complementary rather than mutually exclusive modalities demands proficiency in pharmacologic management. Within such a construct, the likelihood increases that patients will continue to utilize opioid analgesics even after an invasive procedure, albeit with dose titration. Careful dose adjustments are perhaps most essential in the event of a highly successful procedure: reduction in the dose of systemic opioids is often necessary to circumvent toxicity when abrupt reductions in pain result in unopposed opioid effects, while downward titration must be tempered to avoid symptoms of physical withdrawal (abstinence).

**Symptom Control and Quality of Life**

Another lesson from the hospice movement relates to the identification of quality of life as an end point of treatment. A focus on quality of life is also a hallmark of palliative care, a discipline that grew out of hospice and which has achieved the status of a distinct medical subspeciality in some countries. Quality of life refers to the patient's overall functional status and sense of well being, and includes a focus on physical, but also psychological and spiritual aspects of the person.30 More than an abstract concept, quality of life has been the subject of considerable research that has included the development of valid measurement tools.

Studies documenting the frequency of symptoms in patients with cancer have demonstrated that pain is one of the most common problems, present in about 2/3 of patients overall (up to 25% of those with early disease and up to 90% in the setting of advanced disease). Other symptoms are, however, highly prevalent (see Table 1) and if unrelieved result in high levels of distress among patients and family members. If other symptoms are not addressed, even successful pain management will fail to impact on quality of life in a meaningful way. Palliative care experts recommend bringing the same intensity of effort to bear on treating other symptoms as is applied to the management of pain.21, Management of other symptoms is predominantly pharmacologic, and principles governing such management have been advanced in the same way as have guidelines for pain management.21,38 Treatment is often relatively straightforward. It is noteworthy, however that recent developments have suggested therapeutic alternatives for symptoms resistant to standard approaches as well as to symptoms previously viewed as irremediable like cognitive failure, dyspnea, anorexia and weight loss.

Expertise in the management of related symptoms and toxicities is essential for the anesthesiologist-subspecialist, and is facilitated by their expertise in clinical pharmacology. The dictum that treatment with opioids should proceed until pain is controlled or unpleasant side effects supervene implies that significant side effects will be present in a high proportion of patients referred for consultation. Treatment modalities instituted by the anesthesiologist to control pain may produce new symptoms or exacerbate preexisting symptoms. The anesthesiologist's involvement
in overall symptom management is fundamental to the conceptualization of their role as a consultant rather than a technician, or the "doctor at the other end of the needle."

**Trends in Pharmacologic Management**

Pharmacologic management is considered the first line of therapy for patients with cancer pain and when properly applied results in adequate analgesia in the majority of cases. Treatment is effective in adults and children and across different cultures. The analgesia that is associated with systemically administered medications is titratable and suitable for pain that is multifocal and/or progressive. Effects and side effects are reversible, and widespread implementation does not depend on sophisticated technology or scarce resources.27

**Interindividual Variability**

Cancer pain, and indeed pain in general, is characterized by interindividual variability that is manifest in multiple ways. For example, not all bone metastases result in pain. Even when pain is present, self-report varies dramatically as do responses to therapy. The regular administration of an aspirin-like drug may suffice for some patients, while others will require treatment with morphine or another potent opioid. In the latter group, even patients with similar disease characteristics vary dramatically in their analgesic requirements to the extent that daily doses of morphine may need to be dispensed in milligrams for some patients and grams for others.6 Even in the same patient, one standard opioid analgesic may produce dose-limiting side effects, while a pharmacologically similar drug may be tolerated without difficulty due to incomplete cross tolerance. This high degree of interindividual variability mandates careful assessment, and even with the likelihood of favorable treatment outcomes, makes cancer pain management a demanding and time-intensive endeavor.

**Nonopioid Analgesics**

A detailed description of pharmacologic properties and clinical use of these agents is beyond the scope of this paper, but is available elsewhere.11,28,43.

**Nonsteroidal Anti-inflammatory Drugs**

The nonsteroidal anti-inflammatory drugs (NSAIDs) are indicated for mild pain, and combined with stronger analgesics, for moderate to severe pain.11, The NSAIDs are particularly effective for pain of bony metastatic origin, as well as for pain associated with inflammation, due to their inhibitory effects on prostaglandin synthetase (cyclooxygenase),46 an enzyme involved in prostaglandin synthesis. Regular (around-the-clock or a-t-c) administration is most effective. Gastrointestinal, hematologic and renal toxicity may occur, as well as masking of fever, a particular concern in patients with reduced marrow function. In contrast to opioids, the use of the NSAIDs is associated with a ceiling effect, above which dose escalations do not result in enhanced analgesia. The ceiling dose in a given individual may differ from the recommended dose by up to two-fold though, and as a result some dose-titration may still be indicated. Selection is based on the patient's prior experience, minor differences in toxicity, physician experience, schedule and expense.43,50

**Coanalgesics/Analgesic Adjuvants**

The so-called "adjuvant analgesics or coanalgesics" enhance opioid-mediated analgesia, reduce opioid-mediated toxicity or help control other symptoms of cancer. They are a heterogeneous group of medications developed for purposes other than relief of pain, but subsequently determined to have a complementary role in pain management. Of drugs with purported coanalgesic properties, evidence most strongly supports the clinical use of selected antidepressants, anticonvulsants, oral local anesthetics and corticosteroids (see references for details).

**Coanalgesics versus Opioids**

In contrast to the opioids, which are relatively useful for all types of pain, the coanalgesics are indicated only in specific settings, eg: antidepressants,52,.., anticonvulsants,52,.., and oral local anesthetics,43,.., for neuropathic pain and corticosteroids,66 for pain associated with inflammation and peritumoral edema. The dose-response relationship for these drugs and the opioids differs in important ways. The administration of a sufficient dose of an opioid invariably results in some degree of analgesia, which increases linearly with the dose in a close temporal relationship to each administration. Depending on the underlying pain mechanism and other more obscure factors, administration of the coanalgesics may or may not result in analgesia. The onset of analgesia may be delayed by days or even weeks after initiating therapy, and the quality of analgesia is less closely linked to dose increases. As a result, serial trials of each class of coanalgesics, and even of different agents within the same class are indicated.

**Opioid Analgesics**

Oral analgesics are the mainstay of therapy for patients with cancer pain, and are reported to control pain in 70-90% of patients when they are prescribed in accordance with contemporary guidelines.11,23,27,28 The recent introduction of transdermal and oral transmucosal fentanyl provides alternative means to control pain noninvasively.. Pain control can be achieved in a high proportion of remaining patients when opioids are administered parenterally (subcutaneously or intravenously) regionally (intraspinally or intraventricularly) and when
these techniques are combined with other more invasive approaches. The World Health Organization (see Figure 1) has adopted a "ladder" approach to cancer pain management that relies on the administration of oral agents.10 Similar guidelines have been promulgated by the American Pain Society11 and other authorities.27,28

Addiction Redefined
Despite widespread use, the opioids are among the most stigmatized classes of medically available drugs. Misconceptions and other nonmedical factors that detract from optimal use have been described (Table 2), and of these, issues related to the potential for habituation predominate.

Tolerance, physical dependence and psychological dependence (addiction), once considered together as part of a single syndrome are increasingly recognized as distinct phenomena (see Table 3). Physical dependence and tolerance are physiologic effects that are almost invariably associated with chronic opioid use, and as such can be conceived of as independent and distinct from addiction. Addiction (psychological dependence) is regarded as a psychologically-mediated disorder with possible genetic influences that occurs only rarely as a consequence of medical use, and then idiosyncratically. Given acceptance of the validity of this construct, physical dependence, tolerance need not be regarded as important impediments to the successful management of cancer pain with opioid analgesics (see discussion below). Addiction exerts only an indirect negative effect, that correlates with the degree to which clinicians overestimate its risk.

Physical dependence, which also occurs with drugs other than the opioids (eg, benzodiazepines), refers to the probability that a state of withdrawal (abstinence syndrome) will occur if drug administration is abruptly discontinued or a sufficient dose of a specific antagonist is administered. If treatment with the opioids should become unnecessary, physical dependence can be readily managed (avoided) by gradually tapering opioid doses (10-25%/day) and avoiding the use of antagonists. Tolerance exists when, over time, an increased dose of a drug is required to achieve a given effect. It is usually first manifest by a decrease in the observed duration of effect of each administered dose. When tolerance is suspected to be responsible for increased reports of pain, it can usually be countered safely and effectively by simply increasing the dose, especially since tolerance also develops to many of the adverse effects of the opioids, notably nausea and sedation.

Addiction is a complex psychobehavioral syndrome characterized by overwhelming involvement in the acquisition and nonmedical use of a substance despite the threat of physiologic and/or psychological harm.7-7 Although it is a rare sequelae of medical exposure and therefore should not markedly influence prescribing habits, the risk of iatrogenic addiction remains a serious concern among practitioners.6

Selecting an Opioid
The mainstay of treatment for cancer pain of moderate to severe intensity is with potent opioid analgesics, which occupy the highest tier of the three step ladder schema (see Figure 1) recommended by the World Health Organization.9 Patients may access this ladder at any level and may be started on potent opioids initially for severe pain. Also of note is that when patients ascend the ladder serially, less potent analgesics should not be automatically eliminated since the NSAIDs may provide additive analgesia and the mild opioids may be useful for breakthrough or incident pain.

The various opioids produce analgesia by similar mechanisms and when administered in comparable doses, the quality of analgesia and spectrum of side effects are similar. However, individuals vary idiosyncratically in their sensitivity to the analgesic effects and toxicity of the various drugs (incomplete cross tolerance), forming the basis for the clinical use of morphine alternatives. Other reasons for selecting alternate opioid preparations and routes include convenience of dosing and patient satisfaction, variable patterns of pain, gastrointestinal dysfunction, the need for concentrated formulations, and prior favorable clinician and patient experience.

Mild Opioids
Traditionally when treatment with the NSAIDs is associated with insufficient relief of pain or is poorly tolerated, the addition of a member of the class of drugs referred to as the "weak opioids" is recommended as an analgesic of intermediate potency.11 Most mild opioids are available only as combination analgesics (with acetaminophen or aspirin), and while there is probably no ceiling dose for the opioid component of these formulations, the number of tablets that can be taken safely is limited by the amount of simple analgesic (aspirin, acetaminophen). Given the lack of a ceiling dose for the opioid component of these preparations, the distinction between so-called weak and potent opioids is somewhat artificial, influenced more on a cultural rather than medical basis, eg: oxycodone has recently been made available in an uncombined form that can be utilized in progressively higher doses to treat even severe pain. One of the most common prescribing errors relates to continuing the use of codeine-like drugs after they are no longer effective, in an ill-advised attempt to avoid prescribing more potent opioids which are also more highly regulated.6

Propoxyphene, a stereoisomer of methadone has relatively few indications for the management of cancer pain since it is only about 1/2 to 1/3 as potent as codeine6 and is not more effective than aspirin or acetaminophen. Although
codeine is considered the prototypical drug of this class, its emetogenic and constipating effects are disproportionate to its relatively weak analgesic properties. Oxycodone is up to 7.7 times more potent than codeine, and, of this class of drugs, is preferred by many authorities. The potency of hydrocodone and dihydrocodeine lies between that of codeine and oxycodone. They are typically available as combination products and their use may be less regulated than that of oxycodone in some clinical settings.

**Potent Opioids: Morphine**

Morphine remains the standard of reference to which other analgesics are commonly compared. The pharmacokinetic and pharmacodynamic characteristics of a single 10 mg dose of morphine administered intramuscularly forms the basis of most tables and charts compiled to describe the relative characteristics of the opioids (see Table 4). Despite widespread use and extensive research, misconceptions about the use of morphine for chronic pain management continue to interfere with its optimal use (see Table 2).

Morphine is readily absorbed from the gastrointestinal tract and is metabolized in the liver. With chronic use, about 1/3 of the orally administered dose ultimately exerts an analgesic effect (oral bioavailability of 3:1). This is in contrast to the 6:1 parenteral:oral ratio determined from single dose studies for acute pain. Since parenterally administered drug is not subject to this first-pass effect, clinicians may incorrectly perceive parenterally administered opioids as more effective than opioids administered orally. Recent research has focused on the role of morphine metabolites, once thought to be inactive. Morphine-3-glucuronide has been postulated to antagonize opioid analgesia, while morphine-6-glucuronide appears to possess potent analgesic properties and may induce persistent nausea and sedation, especially in the presence of altered renal function. The clinical relevance of these metabolites is currently uncertain.

Morphine is available in a variety of formulations and is appropriate for administration by a variety of routes. The most important distinctions are between (1) so-called "immediate release preparations" (MSIR, Roxanol) which have a short latency to effect (about 30 min) and short duration (2-4 hr) and are usually administered every 4 or 3 hours and (2) "controlled release preparations" (MS Contin, Oramorph) which have a longer latency to effect and duration, and as a result are usually administered every twelve or sometimes eight hours.

**Practical Use of Oral Morphine**

Most patients will require simultaneous treatment with two different formulations of an opioid: a long-acting (basal) analgesic administered around-the-clock (a-t-c) and a short acting analgesic, administered as needed (prn). This schema is analogous to the treatment of diabetes mellitus with long acting (NPH) and short acting (regular) formulations of insulin concurrently.

### Basal (around-the-clock) Analgesia

Since most oncologic pain is constant and unremitting, a time-contingent (a-t-c) schedule for the administration of analgesics is preferable to symptom-contingent (prn) administration. This strategy promotes consistent therapeutic plasma levels and avoids "roller coaster" or sine wave kinetics and dynamics characterized by alternating bouts of pain and toxicity (see Figure 2). If analgesics are withheld until pain becomes severe, sympathetic arousal occurs and even potent analgesics may be ineffective. Prolonged prn administration may lead to the establishment of a pattern of anticipation and memory of pain that predisposes to persistent suffering even after a more regular administration of analgesics has been instituted (see Figure 2). Basal analgesia is usually provided by the administration of controlled release preparations of oral morphine every 12 or 8 hours, or alternatively with transdermal fentanyl, methadone or levorphanol (see below).

### Supplemental (prn) Analgesia

In addition to the above regimen, potent short-acting opioids with minimal potential for accumulation (immediate release morphine, hydromorphone, oxycodone) are generally made available on an as-needed basis, usually at intervals of two to four hours for exacerbations of pain. Such exacerbations, referred to as breakthrough pain may be spontaneous, related to specific activities (incident pain) or, if the dose of the basal analgesic is insufficient, may occur regularly just prior to the next scheduled dose (end of dose failure). When incident pain has been identified, patients should be instructed to utilize rescue doses prior to activity, and in the case of end of dose failure, the dose of long acting analgesic should be raised. When frequent use of these rescue doses or escape doses is observed, the dose of basal analgesic should be increased accordingly. In such cases, relatively tolerant patients generally tolerate increments of 25-50% or more of their basal dose readily. Initiating Therapy Since dose response and side effects vary widely based on a number of physiologic and behavioral factors (eg: age, previous drug history, extent of disease, etc), therapy should be individualized to suit the patient's needs. Effective doses often dramatically exceed guidelines recommended in standard texts (10 mg I.M., 30 mg p.o.), which for the most part are derived from experience with acute or postoperative pain in opioid naive patients. Treatment with oral morphine can be started several ways. In cases of severe pain it may be desirable to
initiate therapy with parenteral morphine which can later be converted to an oral drug regimen using a 3:1 ratio. More commonly, immediate release oral morphine is administered every three to four hours to determine opioid requirements, following which the sum of the daily dose is halved and administered as a controlled release preparation and supplemented by rescue doses of immediate release morphine, each aliquot of which should equal 1/6 to 1/10 of the 24 hour dose. Alternatively, treatment can be initiated with an empirically selected dose of controlled release morphine, supplemented by appropriate doses of immediate release morphine. Regardless of the regimen that is selected, low starting doses with rapid upward titration are preferred to limit the frequency of side effects and enhance compliance.

Dose Titration
The correct dose of morphine (or a morphine-line drug) for the management of cancer pain is the dose that effectively relieves the pain without inducing intolerable side effects. Daily doses of morphine required to adequately relieve cancer pain may vary from 60 to 3000 mg in divided doses. There is no ceiling effect for effect, i.e., an increase in the dose will always produce a concomitant increase in pain relief. The starting dose is gradually and steadily titrated upward until either pain control is achieved or side effects occur. If dose increases result in worsening side effects and only small increments in analgesia, the pain syndrome may be relatively opioid-resistant (eg: neuropathic pain or movement-related incident pain). Relatively opioid-resistant pain may require alterative therapeutic approaches.

Side Effects and their Management
Treatment with the opioids may be associated with side effects although in many cases these are transient and in most cases, manageable. Prompt identification, assessment and management of side effects is a cornerstone to treatment. Adverse effects are often perceived of as barriers to the provision of analgesics in doses required to relieve pain effectively (dose-limiting side effects). Most drug-related side effects can be effectively relieved with careful management, but the same attention and skill required to tailor a pain management program needs to be applied to selecting and titrating drugs to minimize the impact of side effects. Patient education is essential to ensure the best outcome and to avoid confusion between manageable side effects and allergy.

A detailed account of the management of opioid side effects is beyond the scope of this article, and is available elsewhere. The potential for side effects should be carefully explained and patients should be encouraged to report problems as they occur. Constipation is almost invariable and requires prophylactic and continued management with laxatives on a "sliding scale" regimen that provides successively stronger laxatives until a regular bowel habit ensues. The clinician should monitor for the presence of bowel obstruction and fecal impaction. Transient nausea and sedation are relatively common when opioid therapy is initiated, but with continued use, usually resolve within a few days to one week. Patients should be reassured and encouraged to adhere to their prescribed regimen of analgesics while symptomatic treatment is instituted and, as tolerance to these effects develops, later tapered.

Reversible central nervous system (CNS) changes associated with opioid therapy range from mild sedation to somnolence, confusion and delirium. Mild cognitive dysfunction is relatively common but usually manageable, while severe CNS toxicity can usually be avoided. Toxicity occurs most commonly after the initiation of treatment or a dose escalation, and is usually transient. Sudden cognitive changes in patients taking opioids chronically are unlikely to be related to opioid therapy, and other potential causes such as brain metastases or electrolyte disturbances should be considered. Sedation is most likely to emerge as a dose-limiting side effect in the elderly and in patients with relatively opioid-resistant pain problems (incident pain, bone metastases, nerve injury). Sedation can often be minimized by initiating opioid therapy at low doses and titrating upwards gradually. Persistent sedative effects can usually be managed by initiating symptomatic treatment with a psychostimulant (methylphenidate or dextroamphetamine), instituting trials of an alternate opioid or an adjuvant analgesic or consideration of an alternate therapeutic modality such as a nerve block or percutaneous cordotomy.

Other Potent Opioids
The pharmacokinetics and pharmacodynamics of other opioid drugs are similar to those noted for morphine, and are described in detail elsewhere. A formulation of transdermal fentanyl has recently been introduced which, once treatment has been established provides relatively steady plasma levels for up to 72 hours following a single application of a 25, 50, 75 or 100 µg/hr patch. The patch's design effectively converts fentanyl to a long acting agent, although consistent, near-peak levels are not obtained for a period of 12-18 hours after the first application and effects persist for 12-18 hours after system removal. Although a useful alternative for maintaining around-the-clock basal analgesia, because of its long latency to effect, transdermal fentanyl is not recommended when rapid titration is required for unstable pain. Methadone is equipotent with morphine when administered intramuscularly, and is slightly more potent when administered orally. It possesses a long and variable half life (13-51 hrs) that may lead to drug accumulation,
especially in patients who are elderly or who have renal failure. Although inexpensive, most authorities recommend its use only as a second line drug and then call for careful monitoring during the initiation of therapy and after dose increases. Treatment may best be initiated by prn administration until steady state is achieved, following which the interval between a-t-c administration may vary between 4 hr and 12 hr. Levorphanol (Levodromoran) resembles methadone, in that due to its relatively long half life (11 hrs), accumulation may occur, dosing intervals may vary from 4-8 or even 12 hrs, and as a result the same precautions described for methadone apply to its use. A parenteral dose of 2 mg and oral dose of 4 mg is usually equianalgesic to 10 mg of parenteral morphine.

Hydromorphone (Dilaudid) is available in a variety of formulations and can be administered by the oral, rectal, subcutaneous and intravenous routes. It is 7-8 times more potent than morphine when administered parenterally and enterally is about four times as potent as oral morphine (parenteral to oral dose ratio of about 5:1).98 Administered by either route, it’s latency to effect and duration are relatively short (about 30 minutes and 2-4 hrs respectively). The main uses of hydromorphone are for subcutaneous infusions (in view of its solubility of up to 200 mg/ml), oral breakthrough dosing and in patients who are intolerant to morphine.

**Drugs to be Avoided**

Meperidine, although extensively used for postoperative pain, is not recommended for chronic administration. Its oral bioavailability is relatively low (4:1) and its duration of action is relatively short (2-3 hr). The most serious drawback to chronic administration is the potential for accumulation of normeperidine, a toxic metabolite with a long half life that may cause tremors, myoclonus and seizures, especially in patients with renal failure. Brompton’s cocktail one of the first preparations of an oral opioid to gain clinical acceptance is now used only infrequently. Developed at Brompton's Chest Hospital in the United Kingdom it consisted of a mixture of morphine hydrochloride (or heroin), cocaine hydrochloride, alcohol, syrup and chloroform water. In blinded trials it has not been shown to produce analgesia that is superior to an oral opioid administered alone,25 and its use should be discouraged because of the problems associated with titrating fixed dose combinations. Likewise, no advantage has been demonstrated for treatment with heroin, although it is still sometimes used in the United Kingdom and Canada, predominantly for subcutaneous infusions in virtue of its high solubility. Opioids with mixed agonist/antagonist activity and partial agonists are not usually recommended for the treatment of chronic cancer pain.10,11,28 Differential binding to opioid receptor sites, which may confer favorable properties for acute pain management are responsible for a relatively high incidence of psychotomimetic effects, and there is usually a ceiling dose above which further dose increases are not associated with additional analgesia. Patients should not be treated concurrently with a pure agonist drug, and conversion from treatment with one class of drugs to another should be performed only cautiously because of the risk of precipitating a withdrawal reaction.

**Alternate Routes of Administration**

Between 1/3 and 2/3 of patients will benefit from at least the transient use of an alternate route sometime before death. There is no evidence that parenteral administration produces superior analgesia to oral administration, so treatment should be reserved for conditions that render oral administration unreliable, such as weakness, dry mouth, dysphagia, nausea, vomiting, malabsorption or obstruction. Alternate routes may also be considered when an impractical numbers of tablets must be ingested or, acutely when rapid induction of analgesia is required to treat a pain emergency.

Rectal Administration is reliable and effective, but is usually only considered practical for short term use,. A continuous subcutaneous infusion (CSCI) or continuous intravenous infusion (CII) is usually instituted when parenteral opioids need to be administered chronically.72,108,110, With adequate home care support, treatment can be initiated and maintained safely and conveniently without hospitalization. An appropriate home infusion device should be flow-calibrated, portable, battery-driven, inexpensively-leased, easily-taught, suitable for the addition of patient controlled analgesia and equipped with alarms.

Except in selected circumstances (pre-existing indwelling catheter, severe cachexia, pain emergencies) subcutaneous administration is preferred to intravenous administration because it is easier to maintain in the home and is as reliable as intravenous administration.110 Absorption of subcutaneously administered opioids is rapid and steady state plasma levels are generally approached within one hour. Morphine and hydromorphone are most commonly employed for subcutaneous infusions, and should ideally be concentrated to permit infusion at volumes of under 1-2 ml/hr in order to minimize tissue irritation. The interested reader is referred elsewhere for a detailed commentary on instituting and maintaining therapy.72,108,110,111

Investigational Routes Morphine elixir has been successfully administered by the buccal and sublingual routes in preterminal patients for short periods of time. In addition, a preparation of transmucosal fentanyl lozenge has recently been approved for preoperative sedation and is now in clinical trials for the treatment of cancer pain. Preliminary results of treatment with opioids
by these and other routes (intranasal, inhalatory, transdermal iontophoretic, vaginal and stomal) are described elsewhere.\textsuperscript{110}

Invasive and Procedural Approaches This review has emphasized the importance of pharmacotherapy as a means to ensure the anesthesiologist's continued role in the management of cancer pain. Ready access to anesthetic-based interventions remains an essential component of comprehensive cancer pain management, although they are best regarded as a component of a therapeutic matrix that includes antitumor therapy, various pharmacologic strategies, neurosurgical and neuroaugmentative procedures, behavioral and psychiatric approaches. The role of diagnostic local anesthetic blocks, therapeutic neurolysis and regional opioid analgesia is amply reviewed elsewhere. These modalities remain an essential focus for the anesthesiologist with specialized training in pain management, and when applied judiciously, are invaluable in selected populations of patients. While this author has argued for the need of anesthesiologists to develop and maintain expertise in pharmacologic management, the ubiquitous application of these approaches engenders a parallel demand for a renewed emphasis on training and teaching classic anesthetic techniques that might otherwise become obscure. While more outcome data regarding the timing of invasive approaches and their respective indications for various populations of patients is needed, their role for patients with otherwise intractable pain remains unquestioned.

\textbf{Conclusion}

Interest in cancer pain management on the part of medical professionals, the lay public, professional societies and governmental and quasi-governmental institutions has intensified considerably in recent years. There is a trend toward emphasis of the central role of pharmacotherapy due to its overall safety, efficacy, simplicity and applicability. The principles outlined here are readily instituted and should constitute an essential and fundamental component of the anesthesiologist-pain specialist's armamentarium, complementing rather than replacing established expertise in instituting invasive approaches.
ACUTE OXYGEN TREATMENT

Introduction

Oxygen has been used in clinical practice for more than 200 years. It is probably the most widely prescribed medication in pre-hospital and hospital environments. If appropriately used it is life-saving and part of first-line treatment in many critical conditions. It is important that oxygen not only reaches the lungs but is delivered to the tissues. Therefore a good cardiac output, circulation and haemoglobin is vital and is why attention to the circulation is an early part of initial resuscitation (The physiology of oxygen delivery, Update in Anaesthesia 1999;10:3). As with any drug, oxygen should be used when indicated, in appropriate dosage (concentration), and correctly administered for a planned duration.

Oxygen manufacture and storage

When cooled to very low temperatures gases change to either solids, (carbon dioxide), or liquids (oxygen and nitrogen). Oxygen has to be cooled to below -118°C to change to a liquid. When the gas changes form to a liquid, it occupies a much smaller volume. Therefore when a small volume of liquid oxygen is warmed it will make a very large volume of oxygen gas. Oxygen can be stored as either a gas in cylinders or as a liquid in a special container. In the liquid form, a very large quantity of oxygen can be transported or stored in a low volume, although there are problems in keeping the liquid cold as explained below.

Vacuum Insulated Evaporator (VIE). A VIE is a container designed to store liquid oxygen. It has to be designed to allow the liquid oxygen inside to remain very cold. It consists of two layers, where the outer carbon steel shell is separated by a vacuum from an inner stainless steel shell, which contains the oxygen (figure 1). The oxygen temperature inside is about -170°C and the container is pressurised to 10.5 atmospheres (10.5 bar). Gaseous oxygen above the liquid is passed through the super heater to raise the temperature to ambient (outside) levels. It then flows into the hospital pipeline system giving a continuous supply of piped oxygen to outlets on the wards and in theatre. Heat is always able to get into the container and provides the energy to evaporate the liquid oxygen, changing it into oxygen gas which is continuously drawn off into the pipeline system. This escape of gas into the pipeline system prevents the pressure inside the container from rising. If the pressure rises too much (above 17 bar), oxygen is allowed to escape via a safety valve into the atmosphere.

In contrast, if the pressure inside the container falls because of heavy demand in the hospital for oxygen, liquid oxygen can be withdrawn, passed through the evaporator and returned to the VIE in the gaseous form to restore the pressure. The amount of oxygen available in the container is estimated by weighing the container with an in-built device.

The VIE system is used in large hospitals which have a pipeline system, and where liquid oxygen can be supplied by road tanker.

Oxygen cylinders. Oxygen can be stored under pressure in cylinders made of molybdenum steel. Cylinders may be combined to form a bank attached to a manifold. The advantages of combining large cylinders into a bank include a reduction in cost, transportation and constant change of exhausted cylinders. Oxygen cylinders come in several sizes in UK oxygen cylinders are black with white shoulders. The pressure inside at 15°C is 137 bar.

Oxygen concentrators An oxygen concentrator is a device which extracts oxygen from atmospheric air using canisters of zeolite. Nitrogen is filtered out and oxygen produced. The function and successful economics were described in detail. (Oxygen concentrators for district hospitals, Update in Anaesthesia 1999;10:11). When ether is used, the oxygen concentrator should be positioned 1.5m above the floor.

Hypoxia

Hypoxaemia is when the oxygen tension in arterial blood is less than 80mmHg (10.6kPa). Hypoxia is a deficiency of oxygen at the tissue level. Traditionally, hypoxia has been divided into 4 types.

Hypoxic hypoxia in which oxygen tension of arterial blood is reduced
Anaemic hypoxia in which the arterial oxygen tension is normal but the amount of haemoglobin(Hb) available to carry oxygen is reduced.
Stagnant or ischaemic hypoxia in which blood flow to the tissues is so low that oxygen is not delivered to the tissues despite normal arterial oxygen tension and Hb concentration.
Histotoxic hypoxia in which oxygen is delivered to the tissues but a toxic agent prevents the cells using the oxygen.

Recognition of hypoxia. Recognition of tissue hypoxia is not always easy as there are a number of different signs and symptoms. Clinical signs and symptoms include:
Altered mental status (agitation, confusion, drowsiness, coma)
Cyanosis
Dyspnoea, tachypnoea or hypoventilation
Arrhythmias
Peripheral vasoconstriction often with sweaty extremities
Systemic hypotension or hypertension depending on the underlying diagnosis
Nausea, vomiting and other gastrointestinal disturbance

Cyanosis means blueness of the tissues and is due to an excessive amount of deoxygenated Hb in the peripheral blood vessels. Cyanosis appears whenever the arterial blood contains more than 1.5 grams of deoxygenated Hb in each 100 ml of blood (normal Hb 15 g/100 ml). Cyanosis can often be detected in a patient with a normal haemoglobin level when the oxygen saturation is less than 90%. When the oxygen saturation falls in anaemic patients, cyanosis is often absent.

As the clinical signs are non-specific, the best method of assessing oxygenation is to measure peripheral arterial oxygen saturation (SaO₂ < 95% is abnormal) and oxygen partial pressure in the arterial blood (PaO₂ < 80 mmHg (10.6 kPa). Pulse oximeters and blood gas analysis have become more widespread throughout the world. Hypoxia at tissue level may still exist even when SaO₂ and PaO₂ are within normal limits, if there is a low cardiac output, anaemia or failure of tissues to use oxygen (e.g. cyanide poisoning). In this situation the blood lactate concentration rises due to anaerobic metabolism. Lactate can be measured in some laboratories.

Oxygen delivery systems

Oxygen can be delivered to the patient using different devices. There are two main types of devices; fixed and variable performance masks.

Fixed performance masks ensure that the patient receives a constant inspired oxygen concentration (FiO₂) despite of any changes in minute ventilation. These include:

- Closed or semi-closed anaesthetic breathing systems with a reservoir bag, attached to anaesthetic machine with pressurised gas supply.
- Head boxes for neonates - oxygen is piped into the box at a constant inspired oxygen concentration. Sufficient gas flow is needed to flush CO₂ out.
- HAFOE High Air Flow Oxygen Enrichment Devices e.g Ventimask

HAFOE masks (figure 2) are colour coded and each mask states the flow of oxygen in litres per minute required to achieve a specific inspired oxygen concentration. There are holes which allow entrainment of room air by the Venturi principle. Relatively high flows of oxygen are needed: e.g. 8 l/min to ensure an inspired oxygen concentration of 40% and 15 l/min to ensure an inspired oxygen concentration of 60%. The flows of 2, 4 and 6 l/min will provide 24, 28 and 31% oxygen respectively. The patient breathes a fixed concentration of oxygen enriched air because the gas flow is greater than the peak inspiratory flow rate of the patient. Thus there is minimal dilution from atmospheric air. The high gas flow flushes expired gas from the mask preventing rebreathing.

Teaching Point
HAFOE masks use the Bernoulli effect to draw in or entrain a second gas via a side arm. This is the Venturi principle. Gas flowing through a tube is passed through a constriction or narrowing formed in the tube. The gas increases speed to pass through the narrowing, and therefore gains kinetic energy because of the increased velocity. The total energy of the system must remain the same, thus there has to be a fall in potential energy. The potential energy of a gas is the pressure it exerts. Therefore, if there is a fall in potential energy there will be a fall in pressure at that point. A second gas can be sucked
in or entrained through a side arm into this area of low pressure (figure 3).

Variable performance masks/devices. The second type of oxygen delivery system includes those which deliver a variable concentration of oxygen. The oxygen concentration delivered depends on patient minute ventilation, peak inspiratory flow rate and oxygen flow rate. For example, when a patient is breathing with a low minute ventilation and is given a high oxygen flow, oxygen concentration will be relatively high. If the patient breathes more without an increase in oxygen flow, there will be a fall in inspired oxygen concentration. Using these masks the oxygen concentration is not fixed or accurate, but in most situations a flow rate of 2 l/min provides 25-30% O2 and 4 l/min provides 30-40% O2. Examples of these devices include:

Nasal cannula. These do not increase dead space. Inspiratory oxygen concentration depends on the flow rate. No rebreathing occurs. Nasal catheters, 8FG, can be inserted into the nose as far as the pharynx, so that they can just be observed behind the soft palate. A gas flow of 150 ml/kg/min gives an inspired oxygen concentration of 50% in children less than 2 years. No rebreathing occurs. The same concept can be used in adults and the cannula may be fashioned from any soft tipped fine catheter (a fine nasogastric tube or urinary catheter may be used in emergencies). When using nasal catheters they must be taped securely in place so that they cannot migrate down into the oesophagus. Plastic oxygen masks (figure 4) have a small dead space. The effect of the dead space depends on the patient's minute ventilation and oxygen flow. There is usually a small amount of rebreathing.
Oxygen Therapy

The American College of Chest Physicians and National Heart, Lung and Blood Institute published recommendations for instituting oxygen therapy. These include:

- Cardiac and respiratory arrest (give 100% oxygen)
- Hypoxaemia (PaO2 < 59mmHg (7.8 kPa), SaO2 <90%)
- Systemic hypotension (systolic blood pressure <100mmHg)
- Low cardiac output and metabolic acidosis (bicarbonate <18mmol/l)
- Respiratory distress (respiratory rate > 24/min)
- In anaesthesia, “added oxygen” should be used during and after anaesthesia as previously described, (The physiology of oxygen delivery, Update in Anaesthesia 1999;10:3).

Table 1

<table>
<thead>
<tr>
<th>Patients who do not require controlled oxygen therapy</th>
</tr>
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<tbody>
<tr>
<td>Asthma</td>
</tr>
<tr>
<td>Pneumonia</td>
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<tr>
<td>Bronchiolitis</td>
</tr>
<tr>
<td>Respiratory distress</td>
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<tr>
<td>Cardiac or respiratory arrest</td>
</tr>
<tr>
<td>Pulmonary embolus</td>
</tr>
<tr>
<td>Shock</td>
</tr>
<tr>
<td>septic</td>
</tr>
<tr>
<td>hypovolaemic</td>
</tr>
<tr>
<td>cardiac failure</td>
</tr>
<tr>
<td>myocardial infarction</td>
</tr>
<tr>
<td>Carbon monoxide poisoning</td>
</tr>
</tbody>
</table>

Patients who require controlled oxygen therapy

- Chronic obstructive pulmonary disease with hypoxic drive
- Premature infants

Teaching Point

Patients who can be harmed by high concentrations of oxygen are mentioned because they are encountered only occasionally. MOST patients benefit from uncontrolled oxygen and it should be given freely to those with cardiac or respiratory arrest, those with respiratory distress, asthma or hypotension.
Prescribing oxygen - controlled or uncontrolled?

As with any drug, oxygen should be prescribed. It may be prescribed as controlled oxygen therapy where the concentration is prescribed using a HAFOE device. However oxygen is more commonly prescribed at a recommended flow rate using a variable oxygen administration device - this is known as uncontrolled oxygen therapy.

A small group of patients with chronic obstructive pulmonary disease (COPD) have raised CO2 levels and depend on hypoxia to stimulate respiration (hypoxic respiratory drive). This is in contrast to the normal patient where the blood level of CO2 drives respiration. They have a long history of chest disease, are cyanosed, sleepy, have signs of cor pulmonale but are not breathless. In these patients high dose oxygen can reduce respiration and cause respiratory depression. They will develop increased CO2 retention, respiratory acidosis and subsequently will require mechanical ventilation. These patients should receive carefully controlled oxygen therapy, starting at 24-28%, which is progressively increased, aiming to achieve an arterial oxygen tension, ideally, above 50mmHg (6.6kPa) or an SpO2 of 85-90%. These patients are rarely encountered in anaesthetic practice, but the possibility of this situation should be considered in people with severe COPD. Unfortunately the risk of hypercapnia in patients with severe COPD is often overestimated, resulting in inadequate oxygen therapy and death from hypoxia.

Monitoring of oxygen therapy

Clinical monitoring includes observation of conscious level, respiratory and heart rates, blood pressure, peripheral circulation (capillary refill, normally 1-2 sec.) and cyanosis.

If available, additional monitoring can be provided by blood gas analysis and pulse oximetry. Check arterial oxygen blood tension and saturation before administering oxygen whenever possible. After starting oxygen, blood gases or oximetry should be repeated adjusting inspired oxygen concentration to achieve PaO2 more than 59mmHg (7.8kPa) or SaO2 more than 90%. Oximetry provides continuous monitoring of oxygen saturation and is especially helpful if blood gas analysis is difficult or unavailable.

However in the small group of patients with chronic lung disease who depend on their hypoxic drive, respiratory depression can be detected by seeing the patient become more drowsy and a rise in arterial CO2 level. Note that oxygen saturation will not decrease until a late stage.

Risks of oxygen treatment

Fire - oxygen supports combustion of other fuels. Do not smoke when on oxygen!
Absorption atelectasis. Prolonged administration of high concentrations of oxygen can result in atelectasis particularly at lung bases. It is most common following chest or upper abdominal surgery and in those patients with poor lung function and sputum retention.
Renronatal fibroplasia. High arterial oxygen tensions are a major factor in causing retrolental fibroplasias in neonates, which may result in blindness. The condition is caused by blood vessels growing into the vitreous, which is followed later by fibrosis. The low birth weight very premature infant is at risk up to 44 weeks postconceptional age. The level of PaO2 required to cause retinal damage is not known, but an umbilical PaO2 of 60-90mmHg (8-12kPa) is safe. Some doctors believe that the normal term infant is also at risk and that arterial saturation must not exceed 95%. However if the baby is hypoxic or requires resuscitation, oxygen must be given. Oxygen in normal concentrations is also safe for short periods during anaesthesia.
Patients on chemotherapy. It is recognized that patients who have received bleomycin are at risk of developing pulmonary fibrosis if they are given excessive concentrations of oxygen during and after anaesthesia. In these patients controlled oxygen therapy should be prescribed to maintain SaO2 90-95%.

Teaching Point
The oximeter is a very useful instrument, but the clinician must not forget its limitations. It only measures oxygen saturation and therefore when interpreting the readings the shape and importance of the oxygen saturation curve must be remembered. The curve is flatter when the oxygen saturation is more than
93%. Therefore relatively large increases increase in oxygen tension (PaO2) will cause small increases in saturation. In contrast, when the saturation falls below 90%, the oxygen tension will fall rapidly with falls in oxygen saturation.

Conclusion

Oxygen is widely used across all medical specialities. In many acute situations, it is the first drug to be given and is life saving. It should always be considered along with management of the airway, delivery system, the importance of the circulation, constant monitoring and reassessment of the treatment. Dangers of oxygen therapy should be always remembered but should never prevent oxygen form being given.
EMERGENCY DRUGS IN ANESTHESIOLOGY AND CRITICAL CARE MEDICINE

Alteplase (Activase, TPA, Actilyse, Actiplas, Besopartin, Lysatecrt-UPA) Route: IV
Dosage: For acute myocardial infarction 6 mg IV bolus followed by 54 mg within the first hour, followed by 20 mg/hour for 2 hours for a total dose of 100 mg. Patients <65 kg should receive a total of 65 mg. Alternatively: 15 mg IV bolus then 50 mg IV over 30 minutes, followed by 35 mg over 60 minutes ("front loading" regimen).

Anistreptase (Eminase, Iminase) Route: IV Dosage: For acute myocardial infarction 30 units IV over 2-5 minutes

Atenolol (Tenormin, Atenil, Atenolan, Betatop, and others) Route: IV
Dosage: Following acute myocardial infarction, 5 mg IV over 5 minutes every 10 minutes for a total IV dose of 10 mg. Oral atenolol therapy should be initiated immediately after the second IV bolus with 50 mg, followed by another 50 mg oral dose 12 hours later. Oral maintenance therapy is continued with 100 mg daily for at least 10 days.

Benztropine (Cogentin) Route: IV, IM, PO
Dosage: For acute dystonic reactions secondary to neuroleptic drugs, 1-2 mg IM or IV push.

Calcium Route: IV
Dosage: Magnesium intoxication: 4.5-9 mEq via IV infusion at a rate not to exceed 200 mg/minute. Hypocalcemic tetany: 4.5-16 mEq via IV infusion at a rate not to exceed 200 mg/minute. Calcium channel blocker overdose: 5-10 mL (6.8-13.6 mEq) of 10% calcium chloride or 10-20 mL (4.65-9.3 mEq) of 10% calcium gluconate IV over 5 minutes. Hyperkalemia with secondary cardiac toxicity: 2.25-14 mEq IV. Repeat after 1-2 minutes as necessary.

Dalteparin (Fragmin) Route: SC
Dosage: For patients undergoing abdominal surgery who are at risk of thromboembolic complications 2500 IU SC daily

Diltiazem (Cardizem) Route: IV
Dosage: For atrial fibrillation or flutter or supraventricular tachycardia 0.25 mg/kg IV push over 2 minutes. A second bolus dose of 0.35 mg/kg may be administered after 15 minutes if the response to the first bolus was not adequate. The bolus dose(s) is followed by a continuous IV infusion at an initial rate of 10 mg/hour. The dosage may be increased by 5 mg/hour up to a maximum recommended infusion rate of 15 mg/hour.

Diphenhydramine (Benadryl) Route: IM, IV, PO
Dosage: For acute dystonic reactions secondary to neuroleptic drugs, 10-50 mg (up to 100 mg if required) deep IM or IV push.
For adjunctive treatment of anaphylaxis, 10-50 mg (up to 100 mg if required) deep IM or IV push.

Dopexamine (Dopacard) Route: IV Dosage: Heart failure associated with cardiac surgery 0.5-6 µg/kg/minute.

Enoxaparin (Lovenox) Route: SC
Dosage: For prophylaxis of venous thromboembolism in patients undergoing hip replacement surgery 30 mg SC BID.

Ephedrine Route: IM, SC, IV
Dosage: For hypotension associated with spinal anesthesia, sympathectomy overdose with ganglionic-blocking or antiadrenergic drugs, penile erection during spinal anesthesia for transurethral resection of the prostate. The usual dose is 25-50 mg IM or SC. The IV route (slow IV push) may be used if an immediate effect is required. Alternatively, 10-25 mg may be given slow IV push. Additional doses may be given at 5-10 minute intervals up to a maximum of 150 mg.

Epinephrine (Adrenalin) Route: IV, ET, SC
Dosage: Cardiac arrest (VF/unstable VT, EMD/PEA, asystole): 1 mg IV every 3-5 minutes (AHA guidelines) Respiratory distress or hypersensitivity reactions: 0.3-0.5 mg (0.3-0.5 mL of 1:1,000 solution) IM or SC every 20 minutes to 4 hours or 0.5-1.5 mg (0.1-0.3 mL of 1:200 suspension) every 6 hours. Shock: 1-4 µg/minute by continuous IV infusion.

Heparin Route: IV, SC
Dosage: Venous thrombosis: 75-100 units/kg (approximately 5,000-10,000 units) IV bolus followed by a continuous IV infusion of 1,680 units/hour (use 1,240 units/hour if one or more of the following risk factors for bleeding are present: Recent surgery or stroke (within last 2 weeks), thrombocytopenia, history of peptic ulcer disease, GI hemorrhage, or genitourinary bleeding; other conditions that increase the risk of bleeding (eg. hepatic failure or invasive lines)). Alternatively, a infusion rate of 18 units/kg/hour has also been used. Titrate to maintain APPT in range that corresponds to heparin concentrations (by protamine titration) in the range of 0.2-0.4 units/mL (Hirsh J. Heparin. N Engl J Med 1991;324:1565-74). DVT prophylaxis: 5,000 units SC q 8-12 hours

Ipecac syrup Route: PO
Dosage: 15-30 mL followed by 3-4 glasses of water. Do not administer concurrently with activated charcoal since activated charcoal will adsorb ipecac. If both are indicated, induce vomiting with ipecac first then administer the charcoal.

Ketorolac (Toradol) Route: IM, IV, PO
Dosage: For post-operative pain, 30 mg (IM or IV) or 60 mg IM followed by 15-30 mg IM or IV every 6 hours.

Magnesium sulphate Route: IV, IM
Dosage: Seizure prevention and control in pre-eclampsia or eclampsia: 4-5 gm of 50% solution IM every 4 hours.

Metoprolol (Lopressor, Betaloc, Arbralene, Beprolo, and others) Route: IV, PO
Dosage: Following acute myocardial infarction, 5 mg IV push every 2 minutes for a total IV dose of 15 mg. Oral metoprolol at a dose of 50 mg every 6 hours should be started 15 minutes after the last IV bolus dose and continued for 48 hours. The maintenance dose is 100 mg twice daily for at least 3 months.

Milrinone (Primacor, Corotrop, Corotrope) Route: IV
Dosage: For short term management of congestive heart failure a loading dose of 50 æg/kg is infused IV over 10 minutes followed by a continuous IV infusion in the range of 0.375 to 0.75 æg/kg/minute.

Nalmefene (Revex) Route: IV
Dosage: Postoperative opioid depression: 0.25 æg/kg IV every 2-5 minutes until the desired degree of opiate reversal is attained. Known or suspected opioid overdose: 0.5 mg/70 kg.

Naloxone (Narcan, Narcanti) Route: IV, IM, SC
Dosage: Postoperative opioid depression: 1.0 æg/kg IV every 2-5 minutes until the desired degree of opiate reversal is attained. Known or suspected opioid overdose: 0.4-2 mg IV, may repeat up to 10 mg. Continuous IV infusion at 4-5 æg/kg/minute may be used.

Nicardipine (Cardene) Route: IV
Dosage: For acute hypertensive episodes 5 mg/hour via continuous infusion. This initial dosage may be increased by 2.5 mg/hour every 5-15 minutes up to a total of 15 mg/hour. Nicardipine has been administered in small IV push doses in the range of 1.25-2.5 mg when immediate antihypertensive action is required although this mode of administration is not FDA approved in the U.S.

Nimodipine (Nimotop) Route: PO
Dosage: For subarachnoid hemorrhage begin 60 mg PO every 4 hours within 96 hours of SAH and continue for 21 days.

Phenytoin (Dilantin, Fenantoil, Di-Hydan, Epanutin, Epilan-D, and others) Route: IV
Dosage: For status epilepticus, a loading dose of 15-20 mg/kg (approximately 1 gm) IV at a rate not to exceed 50 mg/minute should be given.

Physostigmine (Antilirium) Route: IM, IV
Dosage: For life-threatening toxicity secondary to anticholinergic agents, 2 mg IM or IV over 2 minutes. A second dose may be given if no reversal has occurred and if cholinergic signs and symptoms are not present.

Rocuronium (Zemuron) Route: IV
Dosage: For intubation, 0.6 mg/kg (approximately 50 mg) IV push. For maintenance of blockade, a continuous infusion of 5-10 æg/kg/minute may be used and titrated to the desired degree of blockade.

Sodium thiosulphate Route: IV
Dosage: For cyanide poisoning, 12.5 gm IV over about 10 minutes. Repeat at one-half the original dose if signs reappear within 24-48 hours.

Streptokinase (Kabikinase, Streptase) Route: IV
Dosage: For acute myocardial infarction 1.5 million units IV over 1 hour

Urokinase (Abbokinase, Alphakinase, Actosolv, Uronase, and others) Route: IV
Dosage: For acute myocardial infarction, 2 million units IV bolus followed by 1 million units over 60 minutes.

Vasopressin (Pitressin) Route: IV
Dosage: For bleeding oesophageal varices (unlabeled use in U.S.), 0.2 units/minute initially. The infusion rate may be increased by 0.2 units/minute every hour if bleeding continues and up to 1 unit/minute, although doses of up to 2 units/minute may be tolerated.

Verapamil Route: IV
Dosage: For Supraventricular tachyarrhythmias 5-10 mg IV push over 2 minutes. A second bolus dose of 10 mg may be administered after 30 minutes if the response to the first bolus was not adequate. Although not an FDA approved route of administration, verapamil has been administered via continuous IV infusion at a rate of 5 mg/hour.

Sensory loss along medial aspect of calf.
MONITORING DURING CAESAREAN SECTION

Introduction
Recommendations for monitoring during Caesarean section (CS) have been developed by the American Board of Anesthesiologists and the Obstetric Anaesthetists Association (OAA) in the UK. The OAA’s recommendations are reproduced in full in Box 1. Not all anaesthetists have access to complex equipment, but every anaesthetist should be aware of the potential problems that may be encountered and make appropriate use of the monitors they do have. The requirements for regional and general anaesthetics are different and so considered separately. All obstetric patients undergoing CS should be positioned with left lateral tilt to avoid aorto-caval compression. (Pharmacology of Inotropes and Vasopressors, Update in Anaesthesia 1999;10:4).

Regional anaesthesia
Most of the monitoring is clinical since awake mothers are excellent monitors of their own physiology. The anaesthetist should be continuously present from the start of anaesthesia to the completion of surgery.

Assessment of analgesia
A major cause of maternal complaint is pain during CS under regional anaesthesia. For CS, a block should extend from S4 to the upper thoracic dermatomes. One common reason for inadequate pain relief is a failure of the block to spread to the sacral dermatomes. Although this happens more frequently with epidural than spinal anaesthesia, whichever technique is used, always test the back of the legs (S2 and S3) to confirm that the sacral dermatomes are blocked before surgery starts.

How high a regional block must extend into the thoracic dermatomes to achieve intraoperative analgesia, remains controversial. Recommendations from T10 to T4 have been made, although the method of testing the block is often unspecified and the need for supplemental analgesics not mentioned. The three most commonly used methods of assessment are:
- loss of temperature sensation
- loss of pinprick sensation
- loss of light touch sensation.

These may differ by as much as 10 dermatomes, with temperature sensation lost first and light touch sensation last. Experimental data suggests that intraoperative analgesia is most reliably predicted by blocking light touch sensation (the hub of a needle lightly applied to the skin) to T5 (just beneath the nipples).

Box 1: Recommendations for monitoring during caesarean section
For operative delivery under regional block
Continuous pulse oximetry, non-invasive blood pressure and continuous ECG during induction, maintenance and recovery.

During general anaesthesia
Continuous inspired oxygen and end-tidal carbon dioxide concentration should be monitored, as well as pulse oximetry, non-invasive blood pressure and ECG.

Haemodynamic consequences of regional anaesthesia
Extensive epidural and spinal blocks cause a temporary sympathectomy which makes the patient susceptible to hypotension. In pregnant women, this is made worse by the uterus compressing the aorta and inferior vena cava (aorto-caval occlusion). Hypotension may develop rapidly. Therefore, blood pressure should be measured at least every two minutes from starting a regional block until delivery. Nausea during onset of a regional block is usually an indication of hypotension.

Blocks above T4 cause a loss of sympathetic innervation to the heart which may be associated with bradycardia particularly if aorto-caval occlusion is present. Because of this continuous monitoring of the pulse is essential.

Respiratory consequences of regional anaesthesia
Pregnant women are prone to hypoxia because of a reduction in functional residual capacity (FRC) of the lungs and an increased oxygen consumption. This is compounded during regional blocks by abdominal and intercostal muscle weakness which causes a further reduction in FRC. Pulse oximetry not only monitors the pulse but also provides a continuous non-invasive monitor of the saturation of arterial haemoglobin. It is simple and accurate; always use it if you can.

When the thoracic dermatomes are blocked, patients often complain of a strange sensation when breathing, usually as they realise that they cannot produce a forceful cough. This is normal and a result of intercostal paralysis and
the patient can be reassured. However difficulty in speaking represents diaphragmatic paralysis developing and needs very careful assessment of the level of block. Further spread of local anaesthetic must be minimised. If hyperbaric local anaesthetic has been used, this can be done by careful elevation of the head and neck. However be prepared to intubate and support these patient's ventilation.

**Unexpected high blocks**

"Total spinals" or very high blocks may follow excessive spread of a deliberate intrathecal injection of local anaesthetic or be caused by an epidural catheter that is misplaced in the subarachnoid space. Misplaced epidural catheters can be detected by attempting to aspirate CSF through the catheter and carefully assessing the effect produced by a test dose. An appropriate test dose will produce detectable changes in sensory and motor function within five minutes of injection if the catheter is in the subarachnoid space and no significant effect if the catheter is in the epidural space.

The spread of deliberate intrathecal injections of hyperbaric (heavy) local anaesthetics can be controlled by keeping the upper thoracic and cervical spine elevated. As spinal blocks sometimes extend very rapidly, you must check the spread of the block within 4 minutes of injection and reposition the patient if necessary.

Symptoms of high blocks are predictable. As the block extends the hands become warm and dry, then loss of hand and arm movement follows. Loss of abduction of the shoulder may be rapidly followed by diaphragmatic paralysis. At the same time sensation is lost over the upper chest, hands, arms, shoulder and neck. If the block extends further, consciousness may be lost and the pupils may become fixed and dilated. However all these signs will reverse provided cardiovascular and respiratory support are provided.

Regional blocks may continue to extend for at least 30 minutes after local anaesthetic has been injected, so the anaesthetist must remain vigilant for symptoms of high blocks even after surgery has started.

**Monitoring the injection of local anaesthetic**

Accidental intravenous injection of local anaesthetics may occur with epidural anaesthesia and although deaths are rare, convulsions occur in 1 in 500 - 9000 patients. This risk can be minimised by carefully aspirating before each injection, by assessing the effect of a small initial test dose of local anaesthetic and by splitting all large doses of local anaesthetics into several small portions. Every dose must be assessed for symptoms of intravenous injection (Table 1) even when previous doses have been uncomplicated.

**Table 1: Symptoms of intravenous injection of local anaesthetic**

<table>
<thead>
<tr>
<th>Symptom</th>
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<tbody>
<tr>
<td>Tingling around the mouth</td>
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<tr>
<td>Tinnitus (ringing in the ears)</td>
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<tr>
<td>Visual disturbance</td>
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<tr>
<td>Confusion</td>
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<tr>
<td>Slurred speech</td>
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<tr>
<td>Altered conscious state</td>
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<tr>
<td>Convulsions</td>
</tr>
<tr>
<td>Coma</td>
</tr>
<tr>
<td>Cardiovascular collapse</td>
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<tr>
<td>Cardiac arrhythmias</td>
</tr>
</tbody>
</table>

**General anaesthesia**

Patients undergoing CS performed under general anaesthesia should be monitored in the same way as with any general anaesthetic. The obstetric anaesthetist should be particularly aware of airway problems and episodes of hyper- or hypotension.

**Monitors of intubation**

Failure of intubation and oxygenation remains one of the commonest causes of anaesthetic related maternal deaths. Confirmation of the correct placement of an endotracheal (ET) tube is crucial. Various monitors are available to help the anaesthetist, but seeing the ET tube pass through the glottis remains the most valuable. However the presence of bilateral breath sounds should always be checked and, when possible, the presence of expired CO2 confirmed. Figure 1 shows ten simple clinical tests of correct placement of a tracheal tube.

The oesophageal detection device is a useful additional monitor. It is cheap and easily constructed using a 50 ml syringe or a self inflating bulb. If a negative pressure is applied by the syringe to a correctly positioned endotracheal (ET) tube, gas can be aspirated because the trachea is supported by rigid cartilage. However if the ET tube is misplaced in the oesophagus and a negative pressure applied, the oesophagus will obstruct the tip of the ET tube and gas cannot be aspirated. (Low Spinal Anaesthesia for Caesarean Section, Update in Anaesthesia 1997;7:7)
### Figure 1: 10 Clinical tests of tracheal intubation

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
<th>Significance</th>
<th>Reliability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Look with laryngoscope</td>
<td>Tube passes between cords</td>
<td>Correct tracheal intubation</td>
<td>Certain</td>
</tr>
<tr>
<td>Listen/feel</td>
<td>Breathing through tube</td>
<td>Correct tracheal intubation</td>
<td>Probable</td>
</tr>
<tr>
<td>Tap sternum</td>
<td>Air comes out through tracheal tube</td>
<td>Correct tracheal intubation</td>
<td>Probable</td>
</tr>
<tr>
<td>Inflate with SIB*</td>
<td>Chest rises &amp; falls</td>
<td>Correct tracheal intubation</td>
<td>Probable</td>
</tr>
<tr>
<td>Pass catheter down inside tube</td>
<td>Patient coughs (if not paralysed)</td>
<td>Correct tracheal intubation</td>
<td>Probable</td>
</tr>
<tr>
<td>Look</td>
<td>Patient remains pink after intubation</td>
<td>Correct tracheal intubation</td>
<td>Probable</td>
</tr>
<tr>
<td>Look</td>
<td>Patient becomes cyanosed after intubation</td>
<td>Oesophageal intubation (REMOVE TUBE)</td>
<td>Certain</td>
</tr>
<tr>
<td>Stethoscope</td>
<td>Air entry at both apices both axillae &amp; both bases</td>
<td>Correct tracheal intubation</td>
<td>Probable</td>
</tr>
<tr>
<td>Stethoscope</td>
<td>Air entry over stomach</td>
<td>Oesophageal intubation (REMOVE TUBE)</td>
<td>Probable</td>
</tr>
</tbody>
</table>

* = self inflating bag

The capnograph or an oesophageal detection device (see above) are the most useful pieces of equipment to confirm intubation.

**Monitors of ventilation**

As with regional anaesthesia the pregnant mother is vulnerable to hypoxia; look at the patient's colour and at movement of the chest wall. If you are ventilating by hand feel for any changes in resistance to ventilation - if you are using a lung ventilator look regularly at and make a note of the inflation pressure. Always use a pulse oximeter if you have one.

**Haemodynamic consequences of general anaesthesia**

Aorto-caval occlusion means that mothers are vulnerable to hypotension, while hypertension may occur with laryngoscopy and surgical stimulus. Pre-eclamptic mothers are particularly vulnerable to hypertension on laryngoscopy. So, as with regional anaesthesia, blood pressure must be measured at least every two minutes until delivery and the pulse continuously.

**Monitors for awareness**

To reduce fetal depression and uterine relaxation, anaesthetists have sometimes used low doses of anaesthetic agents in a paralysed mother during CS. This has resulted in some mothers being awake and in severe pain. No single monitor reliably predicts awareness, although signs of sympathetic stimulation - sweating, tachycardia, hypertension and pupillary dilation - should always be regarded with concern.

The most reliable method of ensuring the mother is asleep, is to give adequate doses of induction agents and an initial overpressure of inhalational agents (ie twice MAC for 5 min, 1.5 times MAC for the next 5 min and 0.8 times MAC thereafter).

**Neuromuscular blockade**

With modern short acting muscle relaxants, reversal of neuromuscular block at the end of caesarean section is rarely a problem. The exception is if the mother has been treated with magnesium sulphate. Magnesium enhances the action of non-depolarising muscle relaxants. So in these patients, assessing neuromuscular function is important, ideally with a nerve stimulator but alternatively clinical methods may be used, such as assessment of hand grip or head lift.
**Fetal monitoring**

Various monitors of fetal condition are available. Fetal heart rate (FHR) monitoring is the most common. The FHR may be recorded intermittently with a stethoscope, by abdominal ultrasound, or with a fetal scalp electrode. A normal FHR has a 95% association with good fetal condition, and a prolonged and continuing bradycardia is almost always associated with severe fetal distress.

During CS, the FHR should be monitored from the start of anaesthesia until abdominal skin preparation especially if the fetus is already distressed. Knowing that the FHR is not critical, may allow time for a regional technique to be used, when otherwise a general anaesthetic might have to be performed. Knowledge of the FHR is also useful if a failed intubation occurs during general anaesthesia. The FHR can influence the decision to either wake the mother and perform a regional technique, or continue surgery with a face mask.

**Special problems**

While the monitoring requirements for uncomplicated Caesarean deliveries are straightforward, additional monitors may be required if other pathologies are present. As haemorrhage, embolism, hypertensive disorders of pregnancy and maternal cardiac conditions are associated with more than 50% of maternal deaths in the UK, these conditions deserve special mention.

**Major haemorrhage**

Major haemorrhage may be life threatening. Whenever major haemorrhage occurs, invasive cardiovascular monitoring should be used if available. This should include hourly urine output measurement, temperature monitoring and central venous pressure and invasive arterial pressure monitoring.

**Embolism**

The triad of hypocapnia, hypoxia and hypotension should alert the anaesthetist to the possibility of an embolism. Air embolism, thromboembolism and amniotic fluid embolism may all occur. Minor air embolism can be detected in almost every caesarean section. However it is extremely unusual for this to have any clinical significance. Thromboembolism causes approximately 25% of UK maternal deaths, but rarely presents during surgery. Perioperatively, amniotic fluid embolism is the greatest risk. If embolism is suspected then invasive cardiovascular monitoring should be considered and the clotting cascade assessed. Amniotic fluid embolism is often associated with a coagulopathy.

**Hypertensive disorders of pregnancy**

Severe pre-eclampsia is associated with a reduced plasma volume, while total body water is increased. Laryngeal oedema may make intubation difficult and hypertensive responses to intubation may be greatly increased. Treatment with magnesium may prolong the action of muscle relaxants. *(Prediction and Management of Difficult Tracheal Intubation, Update in Anaesthesia 1998;9:9)* Renal failure may be present. Monitoring should be tailored to detect these problems and particular consideration given to invasive monitoring of central venous pressure, arterial blood pressure and hourly urine output.

**Maternal cardiac conditions**

Pregnancy stresses the cardiovascular system, particularly at delivery, when large fluid shifts and rapid changes in the pre- and after-load of the heart occur. These changes may be compounded by anaesthesia. Patients with cardiac disease, especially significant shunts or stenotic valvular lesions, are vulnerable to these changes. Some patients will require invasive cardiac monitoring throughout the perioperative period.

**Conclusion**

Caesarean sections are so common that the risks are often ignored. However in a recent survey, 82% of anaesthetic related deaths occurred during Caesarean section. The obstetric anaesthetist can reduce the risk to his patients by careful monitoring. The monitors should be tailored to detecting the problems that may be encountered so that they can be corrected before mother or fetus are harmed.
THE PATIENT WITH HEART DISEASE

Introduction
Over the past six decades, mortality due solely to anaesthesia has decreased from approximately 1 in 1,500 to 1 in 150,000. However, death within 30 days of surgery remains a major issue. In the United Kingdom, over the past ten years, the number of such perioperative deaths has remained fairly constant at approximately 20,000 deaths per annum, of which 9,000 are due to cardiac causes. For each cardiac death there are between 5 and 20 major cardiac complications, such as myocardial infarction, unstable angina, life-threatening arrhythmias, or acute left ventricular failure. Thus, the UK number of cardiac complications is expected to range from 45,000 to 180,000 per annum. These complications occur in patients with a compromised cardiovascular system, most frequently because of underlying coronary artery disease. Indeed, 60% of patients who die within 30 days of surgery have evidence of coronary heart disease. Pre-existing valvular heart disease, hypertensive heart disease, and congestive cardiac failure also play an important role.

Coronary heart disease
Angina causes only a moderate increase in perioperative morbidity provided it is well controlled and medication is continued throughout the perioperative period. Unstable, new and disabling angina are associated with a high post-operative morbidity. In such patients coronary angiography is usually necessary prior to major surgery in order to establish the severity of the disease and optimise treatment; this may include coronary angioplasty (with or without stenting) or coronary artery bypass surgery. When the angina is less severe and coronary bypass surgery is not indicated in its own right, prophylactic coronary artery revascularization may be performed, on occasion, in order to reduce the risk of postoperative myocardial infarction, but only in the face of major surgery. However, where coronary revascularisation is indicated in its own right it should be carried out before non cardiac surgery.

Previous myocardial infarction. Myocardial infarction that has occurred less than three months before surgery is known to be associated with a very high risk of reinfarction. In recent years this risk has become smaller. The time that has elapsed between myocardial infarction and surgery is, therefore, one of risk factors. Cardiologists consider that after uncomplicated myocardial infarction a delay of six weeks is acceptable. This view is endorsed in the American College of Cardiologists and American Heart association (ACC/AHA) guideline. Irrespective of the delay between infarction and surgery, risks remain high in patients presenting for major abdominal or thoracic surgery or for vascular surgery, and in patients who have suffered from acute left ventricular failure at the time of their infarction, exhibit poor left ventricular function, or continue to suffer from angina. Therefore evaluation of left ventricular function is essential especially before major surgery as there is an inverse relationship between left ventricular function and adverse cardiac outcome. This evaluation includes clinical examination and exercise tolerance. Often, however, an objective test is needed as many patients minimise their disability, or are incapable of exercising for other reasons (arthritis, severe intermittent claudication) and, therefore, never "test" their cardiac reserve.

Silent myocardial ischaemia, as detected by ambulatory ECG (Holter) monitoring, is a feature of coronary heart disease. It is observed in patients who are totally asymptomatic (type 1), have suffered previous myocardial infarction (type 2), or suffer from angina (type 3). Up to 80% of ischaemic events are silent. Silent myocardial ischaemia is associated with adverse prognosis. Silent ischaemia occurs in up to 50% of adult surgical patients and is associated with postoperative cardiovascular complications. It is more frequent and more prolonged during the postoperative period. This increased ischaemic burden (expressed as minutes of ischaemia per hour of monitoring) is responsible for the very strong association between postoperative silent ischaemia and adverse cardiac outcome. Silent ischaemia may be caused by cardiovascular instability (tachycardia, hypertension, hypotension), coagulation disorders (microthrombosis and microlysis) and/or postoperative hypoxaemia. A feature of perioperative silent myocardial ischaemia is that it is associated with short- and long-term adverse outcome thereby decreasing the event-free survival at two years from 90% to 76%.

Patients with coronary grafts. Many patients undergo non-cardiac surgery after previous coronary bypass graft operation. The risk of postoperative myocardial infarction is low in this group of patients provided they are not operated on less than six weeks to two months after coronary surgery, and do not have other risk factors such as angina or poor left ventricular function. Hypotension during the perioperative period must be avoided as it may cause thrombosis of the grafts. In some patients, significant increases in left ventricular function are observed after coronary artery bypass graft or after angioplasty and stenting, because previously underperfused myocardium (hibernating myocardium) becomes more contractile after reperfusion.

Patients with previous angioplasty and stenting. Some benefits in terms of risk reduction can be expected in patients who have undergone coronary angioplasty more than three months before elective surgery. If a stent is
Ambulatory ECG monitoring is valuable, however, it is now regarded as inferior to formal exercise testing.

Arterial hypertension is associated with an increase in the cardiovascular morbidity and mortality of anaesthesia and surgery23,24, even though hypertension was not found to be a significant predictor of cardiac complications of anaesthesia and surgery in several indices of cardiac risk in non-cardiac surgery.25,26 In patients diagnosed as hypertensive, and on anti-hypertensive medication, treatment of hypertension should be maintained throughout the perioperative period; often the morning dose of ACE inhibitors is omitted because of the risk of hypotension. However, this policy may cause an increase in the risk of perioperative hypertension.27 Treatment with angiotensin receptor antagonists needs to be stopped the day before surgery because of the risk of refractory hypotension after induction of anaesthesia and during surgery.28 Where hypertension is poorly controlled, management of the patients should follow the principles applicable to untreated hypertension.

In untreated patients, mild hypertension (Stage 1: 140-159/90-99mmHg), does not constitute a major threat. Moderate hypertension (Stage 2: 160-179/100-109mmHg), constitutes a threat especially where it is associated with target organ involvement (coronary, cerebrovascular or renal disease), in which case treatment prior to elective surgery is recommended. Severe hypertension (Stage 3: 180-201/110-119), and marked left ventricular hypertrophy (ECG and/or chest X-ray), increase the risk of complications. Such patients should be treated before surgery; this is also true of patients with malignant hypertension (Stage 4: >210/>120).29

Professor Prys-Roberts, in an editorial published in 200130 took a different view of the management of hypertensive patients, suggesting that in untreated patients, postponement of surgery is unnecessary unless the diastolic pressure exceeds 120mmHg. For treated hypertension, cancellation in order to improve treatment may be justified if the diastolic pressure exceeds 110mmHg. Subsequently, Professor Prys-Roberts, in a letter31, adopted a position that is more in keeping with the generally agreed principles. Similarly, the AHA/ACC guideline suggest that patients with a diastolic blood pressure above 110mmHg should be treated before surgery.6

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Heart failure
Patients with heart failure are at risk of major postoperative cardiac events. Even incipient heart failure is a strong predictor of adverse outcome.25,26,32 The number of patients with heart failure is increasing very rapidly because the mortality of myocardial infarction has been reduced and, therefore, more patients survive with impaired cardiac function.33 Evaluation of cardiac function with echocardiography or radionuclide angiography is very useful because the risk of complications of anaesthesia and surgery is directly related to the severity of ventricular dysfunction. An ejection fraction less than 40% predicts adverse cardiac outcome.34 The patient's drug therapy should be optimized before surgery. In some patients coronary bypass surgery16 or coronary angioplasty and stenting improve left ventricular function to such an extent that even major surgery becomes considerably safer.
An increasing number of patients with heart failure are now receiving beta-blockers. The latter improve their long-term prognosis, especially where carvedilol is used. However, in all studies of beta-blockade in heart failure, treatment is initiated with extremely low doses, with increases in dosage over eight weeks or more.35 Recently the possible value of measuring natriuretic peptides has been emphasised.36 In particular Brain Natriuretic Peptide (BNP) has been found to be elevated in patients with cardiac dysfunction.37 It is a predictor of poor survival.38 Measurement of BNP could be used as a screening test for cardiac dysfunction so that further tests would only be performed in selected patients.

**Anaesthetic management of patients with coronary or hypertensive heart disease**

A major requirement is to avoid haemodynamic changes that may precipitate myocardial ischaemia. Tachycardia increases myocardial oxygen consumption and decreases coronary flow because of the shorter duration of diastole. Hypotension may reduce coronary flow more than myocardial oxygen consumption because of low coronary perfusion pressure. Hypertension can cause increases in oxygen demand that exceed the coronary reserve. This adverse effect is worsened when tachycardia is present. However, many episodes of myocardial ischaemia occur in the absence of marked haemodynamic changes.39 These may be caused by coronary artery spasm, transient spontaneous coronary occlusion (microthrombosis), or coronary steal. The latter may develop during the administration of dilators of the coronary resistance vessels. This has been reported with isoflurane (40). Another important contributor to the safety of these patients is the prevention of post-operative hypoxaemia.13 In high risk patients, invasive monitoring, including monitoring of the pulmonary occluded pressure (pulmonary capillary wedge pressure) is useful for major surgery, particularly vascular surgery of the thoracic or abdominal aorta. Transoesophageal echocardiography (TOE) may be useful for the detection of ischaemia and, more importantly, the assessment of ventricular filling. The new generation of ECG monitors are capable of displaying ST-segment trends, thus allowing better detection of perioperative myocardial ischaemia than visual inspection on an ECG monitor.

A prerequisite in the management of patients with coronary or hypertensive heart disease is to protect the myocardium. The first step is to maintain their treatment throughout the perioperative period. However, not all drugs used in the chronic management of these patients are equally effective in preventing cardiac complications of anaesthesia and surgery. A more active approach to ischaemia prevention has developed.

**Active drug prevention of ischaemia**

Over the past five years it has become clear that the management of surgical patients with coronary heart disease could be improved by the prophylactic administration of drugs in order to decrease oxygen demand, make the circulation more stable, or improve the distribution of coronary blood flow. Drugs having such effects include calcium antagonists, adenosine modulators, alpha2 adrenoceptor agonists, and beta-blockers.

Systematic studies of the perioperative prophylactic administration of calcium antagonists are lacking. However, observational studies do not show patients on calcium antagonists to be protected against silent myocardial ischaemia41,42 even though calcium antagonists cause coronary vasodilatation, relieve exercise-induced coronary vasoconstriction, reduce left ventricular afterload, and improve the oxygen balance.

Adenosine modulation causes a selective augmentation of adenosine levels in tissues under ischaemic conditions but not in the non-ischaemic myocardium. This results in improved left ventricular function, enhanced collateral blood flow, reduced risk of ventricular dysrhythmias, and attenuated risk of stunning. Five trials of the adenosine modulator acadenosine (total of 4,043 patients) were analysed together.43 They showed a significant reduction in myocardial infarction (-27%), stroke (-26%), and cardiac death (-50%). Unfortunately, the development of this agent has been stopped.

Alpha2-adrenoceptor agonists decrease sympathetic activity by a central mechanism, this results in better haemodynamic stability, and decreased risk of silent ischaemia. In addition, there is sedation, and reduction in anaesthetic and opioid requirements. Clonidine has been shown to reduce the risk of perioperative myocardial ischaemia.44 In terms of cardiac outcome, a study of the alpha2-adrenoceptor agonist mivazerol showed significant reductions in cardiac death, myocardial infarction and cardiac death, and myocardial infarction and all causes of death, but only in vascular surgical patients.45 Development of this promising agent has been stopped.

Beta-adrenoceptor blockers are known to reduce myocardial oxygen consumption, decrease the effects of sympathetic activation, and redistribute coronary blood flow. They may reduce overall sympathetic outflow. For more than twenty-five years, beta-blockers have been shown to minimise the risk of perioperative myocardial ischaemia.46,47,48 More importantly, perioperative beta-adrenoceptor blockade has been shown to decrease the incidence of perioperative myocardial infarction.49,50 More recently, atenolol given for one week perioperatively51 was shown to result in lower mortality at two years by comparison with administration of a placebo (9% vs 20%).

In 1997, the American College of Physicians published a guideline for assessing and managing the perioperative risk from coronary artery disease associated with major non-cardiac surgery.52 The important message was that for
all patients, eligibility for beta-blocker use should be determined. Further evidence for beneficial effects of perioperative beta-blockade was obtained by Poldermans and colleagues. They studied patients in whom coronary artery disease had been demonstrated by the presence of reversible ischaemia on dobutamine echocardiography. In their study, prolonged beta-blockade, started a week or more before surgery, was associated with a large reduction in cardiac death (3.4% vs 17% in the control group) and non-fatal myocardial infarction (0% vs 17% in the control group). The efficacy of beta-blockade was impressive. Moreover, as patients were maintained on beta-blockers, their long-term prognosis was also much improved. However, as all patients had reversible ischaemia, they were at a particularly high risk for coronary events. Thus, the efficacy of beta-blockade cannot be extrapolated to patients at risk for, rather than with demonstrable coronary artery disease. However, based on published studies, and the efficacy of beta-blockade in patients with coronary heart disease, beta-blockers seem to be the logical answer to the perioperative drug management of patients with risk factors for, or with, coronary artery disease.

Why are they not used much more frequently? There are perceived risks to beta-blockade such as worsening of conduction disorders or airway obstruction in patients with reactive airway disease. There is also the risk of worsening of left ventricular dysfunction. Though beta-blockers are now used successfully in the treatment of patients with heart failure, their introduction shortly before surgery may not be well tolerated, unless the initial dosage is very low and doses are increased slowly over several weeks.

Before using beta-blockers routinely in all at risk patients, it is necessary to consider that the studies of Mangano and colleagues, and Poldermans and colleagues were carried out in patients admitted to intensive care or high dependency units, not to ordinary wards. In ITU or HDU environments, any adverse effects can be easily prevented or treated. On the ward this may not be the case. Indeed, the most recent ACC/AHA guideline suggests that beta-blockers should be used in high risk patients and not necessarily in all patients at risk for coronary artery disease.

Beta-blockers may still be the safest agents to use. The treatment should be started well ahead of surgery rather than just the day before surgery. More importantly, a prospective study of their safety on the ward is warranted. If they prove to be well tolerated then their use could be greatly increased. Hopefully, this coupled with other measures could reduce substantially the number of cardiac complications of anaesthesia and surgery.

If acute beta-blockade is effective in reducing the risk of cardiac complications of anaesthesia and surgery, it is tempting to conclude that patients on chronic beta-blocker treatment are well protected. This is not the case. The incidence of perioperative silent myocardial ischaemia is not reduced in patients on chronic beta-blockade. Similarly, chronic beta-blockade does not appear to reduce perioperative mortality. Chronic beta-blockade may not offer the same degree of protection as acute beta-blockade because of beta-adrenoceptor up-regulation or other factors. Therefore, such patients must be considered to be at risk, and monitored especially carefully.
Anaesthesia and Chronic Renal Failure

Chronic Renal Failure (CRF) may be caused by primary renal disease or by systemic diseases which also affect the kidney. A decrease in nephron function occurs and can lead to a typical clinical pattern. CRF only becomes biochemically evident when less than 40% of the nephrons are functioning. Dialysis (either peritoneal or haemodialysis) is generally not required until less than 10% of nephrons are functioning. Patients with CRF are more likely to have associated atheroma formation and hypertension.

Preoperative Assessment and Treatment of Medical Problems in Renal Failure

The following factors should be considered when assessing a patient for anaesthesia prior to either an elective or emergency procedure.

Fluid balance In CRF sodium and water excretion is relatively fixed and often reduced. The kidneys can have difficulty handling both large fluid loads and dehydration. The degree of hydration should be assessed in the usual way using skin turgor, examination of the mucous membranes, jugular venous pressure, presence of dependent oedema and presence of pulmonary oedema on auscultation. Invasive measurement of central venous pressure may occasionally be indicated. Many patients on dialysis regimens will know their normal hydrated weight and their fluid allowance per day.

The patient must be normovolaemic prior to surgery. Fluid resuscitation should normally be with normal saline but if there has been blood loss this might also have to be replaced.

Biochemical balance Although numerous biochemical abnormalities can exist and the potassium can be low, the most significant biochemical problems related to severe uncorrected renal disease are hyperkalaemia and acidosis. Hyperkalaemia is defined as a serum potassium of more than 5 mmol/l. ECG changes become apparent at 6-7 mmol/l and immediate treatment is needed if the serum potassium is over 7 mmol/l. ECG changes include tall peaked T waves, shortened QT intervals, widened QRS complexes and loss of P waves. Eventually the QRS complexes merge into the T waves to produce a sine wave pattern. Ventricular fibrillation may occur at serum concentrations over 10 mmol/l.

Methods of treating a high serum potassium in an emergency include:

1. Administration of 0.5ml/kg of 10% calcium gluconate (max 20 ml). This has an immediate but transient stabilising effect on the myocardial cells.
2. 50mls of 50% glucose as an intravenous bolus or infusion. Glucose and insulin will produce an immediate migration of potassium into the cells thus reducing the serum level. Blood glucose levels should be closely monitored but unless the patient is diabetic, endogenous insulin will be secreted and maintain normal glycaemia. Alternatively 5-10 units of soluble insulin may be added to the infusion. Apart from the risk of errors which may occur, the patient may also become hypoglycaemic as secretion of endogenous insulin is also stimulated.
3. Administration of 1-2 mmol/kg sodium bicarbonate intravenously over 5-10 minutes. This provides a large sodium and fluid load which may not be desirable.
4. Nebulised salbutamol 2.5 - 5mg will assist in moving K+ into the cells.

Total body potassium levels can then be reduced:

1. By dialysis.
2. With calcium resonium (0.5 g/kg) 8 hourly either rectally or orally. This takes approximately 12 hours to produce an effect.
3. By the introduction of a low potassium diet.

Acidosis can best be improved by dialysis. Administration of bicarbonate solution should only be considered when the pH is <7.2. Side effects of bicarbonate solutions include hypernatraemia and volume overload.

Cardiovascular status Hypertension may be a primary problem, secondary to chronic salt and water retention or to excess renin production. Blood pressure must be controlled preoperatively. Ischaemic heart disease is more common and should be assessed preoperatively. Pulmonary oedema may occur with fluid overload or with left ventricular failure. Pericarditis can occur in uraemic conditions.

Respiratory function Pulmonary oedema and pleural effusions both cause a decrease in lung compliance, functional residual capacity and increased ventilation/perfusion mismatch. All these increase the likelihood of hypoxia and are best treated by fluid removal with diuretics or dialysis.

Haematological function Chronic anaemia is common in patients with CRF who are not being treated with erythropoeitin and is usually well tolerated. Unless the patient has ischaemic heart disease the haemoglobin level may be maintained at around 7-8 g/dl. Uraemic patients may have a bleeding tendency due to a decrease in platelet adhesion and fragility of the vessel walls.
Gastrointestinal system
Anorexia, nausea, vomiting, bleeding from stress ulceration, diarrhoea and hiccups are all common symptoms. These can exacerbate dehydration. Nutrition is often poor and this can impair wound healing.

Central nervous system
Uraemia causes malaise, fatigue, decreased mental ability and eventually coma. Severe uraemia or fluid or electrolyte imbalance may cause convulsions.

Endocrine system
Hyperparathyroidism leads to demineralisation of bone making patients more susceptible to fractures. Diabetic control may be difficult because of decreased sensitivity to insulin.

Multiple medications
Patients may be taking corticosteroids or other immunosuppressants which cannot be stopped. Other treatments may have been prescribed for associated diseases.

Dialysis regimen
Those patients with end stage renal failure who are maintained on peritoneal dialysis should continue dialysing until they go to theatre. Haemodialysis should be ideally undertaken with minimum heparinisation up to 12 hours prior to elective surgery.

Pharmacology of Anaesthetic Agents in Renal Failure

The excretion of water soluble drugs and their active metabolites will be impaired. For drugs which are renally excreted the half life increases slowly with deteriorating renal function until severe nephron loss at which point the half life increases sharply with further reductions in renal function. Dialysis can only usually replace a small part of the excretory capacity of the healthy kidney.

Induction agents
Their effect is terminated by redistribution. All of these agents are myocardial depressants and should be administered cautiously in patients with heart disease. Muscle relaxants Suxamethonium should be avoided if hyperkalaemia is present.

Some non-depolarising muscle relaxants depend on the kidney for elimination. Atracurium is the agent of choice as it undergoes spontaneous Hoffman degradation at body temperature.

Vecuronium and mivacurium are safe to use in renal failure as only small percentages are excreted renally.

Gallamine should be avoided and pancuronium, alcuronium, pipercuronium, curare and doxacurium should be used with caution. Potentiation of neuromuscular blockade may occur in the presence of a metabolic acidosis, hypokalaemia, hypermagnesaemia, or hypocalcaemia and with medications such as aminoglycosides. Monitor neuromuscular blockade whenever possible.

Opioids
Morphine is metabolised in the liver to morphine-6-glucuronide which has about half the sedative effect of morphine with a markedly prolonged half life. Pethidine is partially metabolised to norpethidine which is less analgesic and has excitatory and convulsant properties. Both of these metabolites may accumulate in renal failure after repeated doses or with infusions. Standard intraoperative use will not usually cause problems. When available, morphine is preferable to pethidine. Fentanyl and alfentanil can be used as normal. Benzodiazepines can be used in renal failure.

Inhalational agents
There is decreased elimination of the fluoride ions which are significant metabolites of enflurane, sevoflurane and methoxyflurane which can worsen renal function, so these inhalational agents should be avoided especially if used at low flows.

Non steroidal anti inflammatory agents (NSAIDS) should be avoided as all decrease renal blood flow and may precipitate complete renal failure.

Conduct of Anaesthesia

Premedication
Oral sedatives such as diazepam or temazepam may be used. H2 antagonists or non particulate antacids (e.g. sodium citrate) should be given if oesophageal reflux is a problem.

Anaesthesia
Venous access may be difficult. If future haemodialysis is planned it is important to preserve AV fistulas and potential fistula sites. Forearm and antecubital veins should be avoided if possible in these patients.

Full monitoring must be established prior to induction of anaesthesia, with special attention being paid to the ECG and blood pressure. The patient should be kept well oxygenated and haemodynamically stable. Hypovolaemia and hypotension worsen renal function therefore blood and other fluid losses should be carefully replaced. If possible the shorter acting sedative agents should be used.

If spinal or epidural anaesthesia is being performed fluid preloading should be kept to a minimum and vasoconstrictors used to maintain the blood pressure. Otherwise postoperative fluid overload may necessitate dialysis.

Postoperatively Postoperative fluid balance must be meticulous and prompt action taken to limit vomiting and replace any fluids lost. Some patients may require haemodialysis for fluid overload postoperatively but this should be delayed if possible as the patient will have to be heparinised. Some patients may become drowsy on relatively low doses of analgesics.

Oxygen (2-3 litres/minute nasally or 3-4 litres/minute via face mask) should be administered for 48 hours after major abdominal or thoracic surgery and 24 hours after intermediate surgery.
Preventing Acute Renal Failure

Previously healthy patients most at risk of developing acute renal tubular necrosis are those with massive haemorrhage, multiple trauma, sepsis, extensive burns and crush injuries, especially if they already have some degree of renal impairment. Renal failure is diagnosed when urine output is persistently <0.5ml/kg/hour or the serum creatinine rises.

The maintenance of normovolaemia and an adequate renal perfusion pressure are the two most important factors in avoiding acute renal failure. The underlying clinical problem should be controlled and treated as far as possible and adequate hydration guided if necessary by central venous pressure measurement. The urine output should be measured hourly and should be maintained above 1 ml/kg/hr.

Only after the patient is well resuscitated with fluid should vasoactive drugs be used to maintain an adequate mean arterial blood pressure for the patient (this will depend on the patients preoperative blood pressure). If the patient becomes oliguric (urine output < 0.5 ml/kg/hr) despite adequate hydration and blood pressure administration of frusemide can be considered, up to 240 mg intravenously over 1 hour. If no diuresis develops further administration of frusemide is useless. Dopamine and mannitol both increase urine output but also increase the oxygen demand of the kidney so frusemide is preferred. Low dose dopamine has not been shown to have any protective effect on the kidney.

All nephrotoxic drugs should be avoided if possible. These include NSAIDS and ACE inhibitors. If aminoglycosides are essential their serum levels must be monitored.

Electrolytes including potassium, sodium and bicarbonate must be measured at least daily during the perioperative period. Adequate calorie intake is essential and must be established as soon as possible postoperatively.

<table>
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<th>Table 1: Preoperative assessment of the patient in chronic renal failure</th>
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<td>Fluid balance</td>
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<td>Biochemistry and acid base status</td>
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<td>Associated illnesses</td>
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<th>Table 2: Common biochemical and haematological abnormalities in chronic renal failure</th>
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<td>Hyper- (or hypo-)kalaemia</td>
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<tr>
<td>Hypo- (or hyper-)natraemia</td>
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<tr>
<td>Hyperphosphataemia</td>
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<td>Hypocalcaemia</td>
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<tr>
<td>Metabolic acidosis</td>
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<td>Normochromic normocytic anaemia</td>
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<th>Table 3: Treatment of the acutely oliguric patient</th>
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<td>Control the underlying cause if known</td>
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<tr>
<td>Ensure the patient is well hydrated using invasive monitoring if necessary</td>
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<td>Ensure the blood pressure is normal or above normal for that patient</td>
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<tr>
<td>After fluid resuscitation try frusemide 240 mg over 1 hour</td>
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<td>Avoid all non-essential nephrotoxic drugs</td>
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<tr>
<td>Adjust doses of renally excreted drugs</td>
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<tr>
<td>Measure sodium, potassium, bicarbonate and urea and/or creatinine twice daily</td>
</tr>
<tr>
<td>Establish low potassium nutrition as soon as possible</td>
</tr>
</tbody>
</table>
ANAESTHESIA AND LIVER DISEASE

Liver disease can vary in severity from sub-clinical to end-stage liver disease (ESLD), with life threatening, multi-organ multi-system failure. Anaesthetic and operative risks are related to the severity of liver dysfunction, so thorough pre-operative assessment is essential for safe peri-operative care. A good understanding of the pathophysiology of liver dysfunction is vital for assessment of operative risk.

PATHOPHYSIOLOGY OF ESLD

While the severity of liver dysfunction may vary widely, a limited review of the pathophysiology of severe disease is appropriate. The range of symptoms depends largely upon whether the disease presentation is acute or chronic. If chronic, features may be superimposed on a background of poor nutrition and chronic ill health. The acute presentations have been subject to re-classification in recent times in order to take into account differing survival rates with medical treatment alone. The first formal definition was proposed by Trey and Davidson who defined fulminant liver failure (FHF) as the appearance of encephalopathy within eight weeks of onset of jaundice. The King's College group now recognises three levels of acute presentation within FHF (Table 1). Contrary to expectation, survival rates are better in the acute and hyper-acute groups on medical management alone. Gimson has written a useful review of fulminant hepatic failure.

Impaired liver function

Impaired liver function gives rise to effects directly attributable to the failing liver itself and also to indirect effects expressed via other organ systems. Effects directly attributable include hypoglycaemia, lactic acidosis, hypermetabolism, azotemia and impaired urea synthesis. Jaundice appears when serum bilirubin exceeds 35 µmol/l and defects in cholesterol metabolism together with intra-hepatic cholestasis may lead to production of poor quality bile and malabsorption of fat and fat-soluble vitamins. There is reduced synthesis of proteins such as albumin, clotting factors, thyroid binding globulin and pseudo-cholinesterase. Impaired hormone biotransformation, reduced production of modulator proteins and reduced protein binding lead to increased circulating levels of hormones such as insulin, thyroxine, T3, aldosterone and oestrogen. Impaired hormone modulation, failure to clear by-products of metabolism, activation of cytokines and release of vasoactive substances from the damaged liver result in pathophysiological changes in many organ systems. These indirect effects include:

Cardiovascular changes

Vasodilatation and vascular shunting are almost invariable in ESLD. Low systemic vascular resistance (SVR) results in high cardiac output and high mixed venous oxygen saturations. Pulmonary hypertension may develop, while portal venous hypertension can lead to varices, variceal bleeding and porto-systemic shunting. Low flow in the portal vein can result in portal venous thrombosis. Variceal bleeding may be life threatening.

Pulmonary changes

Pulmonary problems are both vascular and mechanical. Intra-pulmonary shunt dilatation (hepato-pulmonary syndrome), impaired hypoxic vaso-constriction and ventilation perfusion mismatch lead to arterial desaturation and clubbing if chronic. Pleural effusions together with ascites can cause considerable mechanical embarrassment of respiration and a reduction in functional residual lung capacity.

Electrolytes and Renal

There are numerous causes of renal impairment in liver failure, including hepato-renal syndrome, sepsis and renin-angiotensin activation. Hyponatraemia due to water retention and inhibition of membrane bound Na/K ATPase, hypoalbuminaemia and oedema are common. Saline should be avoided but hypomagnesaemia and hypophosphataemia should be corrected.

Neurological problems

Mechanisms leading to deepening encephalopathy, loss of vascular auto-regulation, cerebral oedema and death are incompletely understood. A number of processes may act in parallel, but can be summarised as the accumulation of neurotoxic compounds penetrating an impaired blood-brain barrier. At the same time, lack of nutrients and substrates may impair brain metabolism and alter neuro-transmitter synthesis. Of particular interest are a group of endogenous benzodiazepine-like substances that are thought to act at a site closely linked to the g- amino butyric acid (GABA) receptor. Drowsiness can be transiently reversed by flumazenil, but not in all cases. Symptoms can occur in chronic as well as in acute disease, may be rapid in onset and may be precipitated by a gastrointestinal bleed, dietary protein overload or sepsis. Somnolence can be exacerbated by sedative drugs and narcotics. Rapid correction of hyponatraemia can lead to osmotic demyelination and central pontine myelinolysis and should be avoided.

Haematological

Anaemia may be the result of nutritional deficiency, toxic bone marrow depression or gastrointestinal bleeding from varices or erosions. Coagulation defects arise from thrombocytopenia, platelet dysfunction and decreased levels of
circulating clotting factors. Clotting factor levels fall because of impaired synthesis, vitamin K malabsorption and intravascular consumption. The short half-life of clotting factors means that INR or Prothrombin Ratio (PTR) can reliably be used to evaluate residual hepatic function.

**Susceptibility to infection**

There may be a wide variety of defects in host defences that can contribute to a substantial risk of sepsis, with up to 80% of patients with FHF developing bacterial sepsis (frequently Gram positive organisms) and 30% fungal sepsis. Clearly, particular attention must be paid to aseptic technique when inserting lines.

**Drug disposition**

There may be considerable derangement of drug handling in the patient with liver dysfunction. Aetiology may influence pharmacokinetics and the nature and extent of hepatocellular damage may alter drug metabolism. Cholestasis will reduce absorption of fat-soluble drugs after oral administration, while other drugs with limited systemic availability due to high hepatic extraction, may achieve high peak plasma concentrations if there is porto-systemic shunting. Compartment changes and altered protein binding will affect volume of distribution, clearance and re-distribution. Patients with liver dysfunction may be particularly sensitive to opiates and benzodiazepines due to altered end-organ sensitivity (see 'Neurological problems' above).

**Table 1: Liver Failure: mode of presentation and survival**

<table>
<thead>
<tr>
<th>Presentation</th>
<th>Encephalopathy</th>
<th>Jaundice (Days)</th>
<th>Survival Rate (medical management only)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chronic</td>
<td>No</td>
<td>*/-</td>
<td>-</td>
</tr>
<tr>
<td>FHF Sub-acute</td>
<td>Yes</td>
<td>29-72</td>
<td>14%</td>
</tr>
<tr>
<td>FHF Acute</td>
<td>Yes</td>
<td>8-28</td>
<td>26%</td>
</tr>
<tr>
<td>FHF Hyper-acute</td>
<td>Yes</td>
<td>&lt;7</td>
<td>36%</td>
</tr>
</tbody>
</table>

**Causes of Liver Failure**

The commonest causes of acute and chronic liver failure (ALF) are listed in Table 2. In the UK, paracetamol poisoning was until two years ago the most frequent cause of FHF. When a change in regulations reduced over-the-counter pack size of paracetamol to a maximum of 8 tablets, the incidence of paracetamol poisoning fell dramatically. Worldwide, by far the major cause of liver disease is viral infection, with Hepatitis B (HBV) and C (HCV) together accounting for 75% of all cases. The natural history of chronic infection with both HBV and HCV includes progression to cirrhosis and an increased risk of developing hepatocellular carcinoma (HCC). Infection with HCV deserves special mention. The nature of HCV replication is such that during the course of a single infection, HCV frequently changes its antigenic signature. As a result of this and of other mechanisms, the virus is able to confuse host immune responses, with the result that 85% of HCV infections become chronic, as opposed to about 5% in the case of HBV. Chronic HCV infection is insidious and it may take up to 15 years for overt signs of liver failure to develop. Not only can apparently stable, asymptomatic patients decompensate acutely as a result of anaesthesia, but they can also represent a significant infection risk for the anaesthetist.

**Risk and severity scoring**

In 1964, Child and Turcotte classified risk for patients with liver cirrhosis undergoing porto-caval anastomosis for management of portal hypertension. Pugh et al at King's College Hospital published a severity scoring system for patients undergoing oesophageal transection for bleeding oesophageal varices. The two systems have been amalgamated and provide a disease severity assessment based on two clinical and three laboratory variables (Table 3).

**Surgery in patients with liver dysfunction**

The Child-Pugh classification is a useful method of staging the progress of liver decompensation. However, despite its surgical pedigree, it is of limited predictive value in anaesthesia and surgery, because Group B and C patients all represent a high perioperative risk. In general surgical practice, only emergency or life-saving procedures should be undertaken in these patients. In a unit where liver transplantation is an option, other procedures can be considered, particularly those intended to 'buy time' until a suitable donor organ is available. Group A patients are lower risk and with sufficient care can be considered as candidates for most types of surgery.

Hepatocellular carcinoma is a recognised complication in those with chronic HBV or HCV infection. Rates are reported to run between 800 and 2000 cases per year per 100,000 chronically infected. Even in Group A patients, operative mortality for hepatic resection of tumour runs at 5%-10%.

Group B/C patients present an extremely high operative risk and surgical procedures in these patients should be avoided if possible. Considerable morbidity and a high mortality rate invariably accompany all but minor surgery. Procedures that might be performed in these patients include:

- Laparotomy for perforation or bleeding - often following previous surgery
• Porto-systemic anastomosis for portal hypertension: includes meso-caval and distal lienorenal anastomosis.
• Encephalopathy is a common post-operative complication.
• Peritoneo-venous shunting for intractable ascites where liver transplantation is not an option.
• Hepatic resection of tumour in Group B/C patients carries an operative mortality of about 50%.

Table 2: Causes of Liver Failure (UK)

<table>
<thead>
<tr>
<th>CHRONIC</th>
<th>ACUTE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infection</td>
<td>Infection</td>
</tr>
<tr>
<td>Viral A-E, Non A-E</td>
<td></td>
</tr>
<tr>
<td>Drugs</td>
<td></td>
</tr>
<tr>
<td>e.g. paracetamol, rifampicin,</td>
<td></td>
</tr>
<tr>
<td>phenytoin, halothane</td>
<td></td>
</tr>
<tr>
<td>Toxins</td>
<td></td>
</tr>
<tr>
<td>Amanita phalloides</td>
<td></td>
</tr>
<tr>
<td>Miscellaneous</td>
<td></td>
</tr>
<tr>
<td>Wilson's Disease</td>
<td></td>
</tr>
<tr>
<td>Fatty liver of pregnancy</td>
<td></td>
</tr>
<tr>
<td>HELLP</td>
<td></td>
</tr>
<tr>
<td>Lymphoma</td>
<td></td>
</tr>
<tr>
<td>Sepsis</td>
<td></td>
</tr>
<tr>
<td>Reye's syndrome</td>
<td></td>
</tr>
<tr>
<td>Heatstroke</td>
<td></td>
</tr>
<tr>
<td>Viral</td>
<td></td>
</tr>
<tr>
<td>Biliary obstruction</td>
<td></td>
</tr>
<tr>
<td>1°: PBC</td>
<td></td>
</tr>
<tr>
<td>2°: Congenital, stone</td>
<td></td>
</tr>
<tr>
<td>Alcohol</td>
<td></td>
</tr>
<tr>
<td>Toxins</td>
<td></td>
</tr>
<tr>
<td>Drugs</td>
<td></td>
</tr>
<tr>
<td>Auto-immune</td>
<td></td>
</tr>
<tr>
<td>Metabolic diseases</td>
<td></td>
</tr>
<tr>
<td>Wilson's disease</td>
<td></td>
</tr>
<tr>
<td>a1-Antitrypsin deficiency</td>
<td></td>
</tr>
<tr>
<td>Veno-occlusive</td>
<td></td>
</tr>
<tr>
<td>Budd-Chiari</td>
<td></td>
</tr>
</tbody>
</table>

Table 3: Child-Pugh Score

<table>
<thead>
<tr>
<th>Clinical or biochemical measurement</th>
<th>Points scored</th>
</tr>
</thead>
<tbody>
<tr>
<td>Encephalopathy grade</td>
<td>None, 1-2, 3-4</td>
</tr>
<tr>
<td>Ascites</td>
<td>Absent, Mild, Moderate, Severe</td>
</tr>
<tr>
<td>Bilirubin (µmol/l)</td>
<td>&lt;35, 36-60, &gt;60</td>
</tr>
<tr>
<td>Albumin (g/l)</td>
<td>&gt;35, 28-35</td>
</tr>
<tr>
<td>PT (secs. prolonged)*</td>
<td>1-4 secs, 4-6 secs</td>
</tr>
<tr>
<td>[INR]</td>
<td>&lt;1.7, 1.7-2.3, &gt;2.3</td>
</tr>
</tbody>
</table>

*Score prothrombin time or INR
Child-Pugh A Score < 6,
Child-Pugh B Score 7-9,
Child-Pugh C Score >10
CLINICAL MANAGEMENT OF DIABETES MELLITUS DURING
ANAESTHESIA AND SURGERY

Introduction
Diabetes is a condition where the cells of the body cannot metabolise sugar properly, due to a total or relative lack of insulin. The body then breaks down its own fat, proteins and glycogen to produce sugar, resulting in high sugar levels in the blood (hyperglycaemia) with excess by-products called ketones being produced by the liver.

There are two main types of diabetes (table 1) which classically affect different age groups. In reality there is a huge overlap between age groups.

<table>
<thead>
<tr>
<th>Table 1. Classification of diabetes mellitus *</th>
</tr>
</thead>
<tbody>
<tr>
<td>Insulin Dependent (Type I)</td>
</tr>
<tr>
<td>Age of onset</td>
</tr>
<tr>
<td>Sixties onwards, occasionally younger</td>
</tr>
<tr>
<td>Pancreas unable to produce insulin (autoimmune disorder)</td>
</tr>
<tr>
<td>Body unable to use insulin properly</td>
</tr>
<tr>
<td>Treatment</td>
</tr>
</tbody>
</table>

* Note. This is a general classification and there is considerable overlap. Obesity is a common cause of Type II- the pancreas cannot make enough insulin for the body size. Diet/oral hypoglycaemics may initially be enough but eventually insulin may be required.

Diabetes causes disease in many organ systems, the severity of which may be related to how long the disease has been present and how well it has been controlled. Damage to small blood vessels (diabetic microangiopathy) and nerves (neuropathy) throughout the body results in many pitfalls for the unwary anaesthetist. The following guidelines should help to identify these problems and cope with them.

Preoperative assessment.
The general preoperative assessment has been reviewed in a previous article. Preoperative Preparation, Update in Anaesthesia 1997;7:3.

Specific problems arise:
Cardiovascular- diabetics are more prone to hypertension, ischaemic heart disease, cerebrovascular disease, myocardial infarction which may be silent and cardiomyopathy. Damage to the nerves controlling the heart and blood vessels (autonomic neuropathy) may result in sudden tachycardia, bradycardia or a tendency to postural hypotension. A history of shortness of breath, palpitations, ankle swelling, tiredness and of course chest pain should therefore be sought and a careful examination for heart failure (distended neck veins, ankle swelling, tender swollen liver, crackles heard on listening to the chest) made. A preoperative ECG should be performed. Heart failure is a very serious risk factor and must be improved before surgery with diuretics. Table 2 describes how to test clinically for autonomic neuropathy.

<table>
<thead>
<tr>
<th>Table 2: Detecting autonomic neuropathy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tests for autonomic neuropathy</td>
</tr>
<tr>
<td>Sympathetic System</td>
</tr>
<tr>
<td>Decrease &lt; 10 mm Hg</td>
</tr>
<tr>
<td>Measure heart rate response to deep breathing</td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>

Note: if above detected, patient at risk of unstable BP, myocardial ischaemia, arrhythmias, gastric reflux and aspiration, inability to maintain body temperature under anaesthesia.

Renal - kidney damage may already be present, often indicated by the presence of protein (albumin) in the urine. Urine infections are common and should be treated aggressively with antibiotics. The diabetic is at risk of acute renal failure and retention postoperatively. Blood electrolyte measurement (if possible) may reveal a raised urea..
and creatinine. If the potassium is high (> 5 mmol/l) then specific measures should be taken to lower it before surgery.

Respiratory - diabetics, especially if obese and smokers, are particularly prone to chest infections. Chest physiotherapy pre and postoperatively are indicated, with nebulised oxygen and regular bronchodilators (salbutamol 2.5-5mg in 5ml saline) if wheeze is heard. A chest X-ray, blood gases and spirometry are the gold standard investigations, but careful repeated clinical assessment will usually reveal when a patient is as good as they are going to get. Non-emergency surgery should be delayed until this point.

Airway - thickening of soft tissues occurs eg ligaments around joints. If the neck is affected there may be difficulty extending the neck, making intubation difficult. To test if the patient is at risk, ask them to bring their hands together as in praying. If they cannot have the fingers of each hand flat against the other hand, then they probably have ligament thickening of the finger joints, and difficult intubation should also be anticipated.

Gastrointestinal - the nerves to the gut wall and sphincters can be damaged. Delayed gastric emptying and increased reflux of acid make them more prone to regurgitation and at risk of aspiration on induction of anaesthesia. A history should be sought of heartburn and acid reflux when lying flat; if present they should have a rapid sequence induction with cricoid pressure, even for elective procedures. If available, prescribe an H2 antagonist and metoclopramide as a premedication. Ranitidine 150mg or cimetidine 400mg plus metoclopramide 10mg orally 2 hours preoperatively to reduce the volume of stomach acid.

Eyes - cataracts are common, as is an abnormal growth of blood vessels inside the eye (retinopathy). The anaesthetist should try to prevent sudden rises in blood pressure that might rupture them, further damaging the eyesight. Ensure an adequate depth of anaesthesia, especially at induction.

Infection - diabetics are prone to getting infections that can upset their sugar control. If possible, delay surgery until these are treated. Wound infections are common. Great care should be paid to aseptic techniques when any procedure is undertaken.

Miscellaneous - diabetes may be caused or worsened by treatment with corticosteroids, thiazide diuretics and the contraceptive pill. Thyroid disease, obesity, pregnancy and even stress can affect diabetic control.

Blood and urine glucose monitoring - meter analysis (most accurate) or reagent strips (which employ a visual colour comparison with a pre-printed chart) are commonly available. It is vital that the instructions are properly followed for whatever method is used. Out-of-date strips will give an inaccurate reading. If strips are cut in half for economy (not recommended), then the unused portion must be carefully stored in a dry place. When using meters, ensure that the testing strips are properly matched for the meter. Remember, false readings could lead to the wrong, even life threatening treatment being given. Strips or tablets can also be used to test the urine for glucose or ketones. The same precautions apply.

Anaesthetic management:

Many of the operations diabetic patients face are a direct result of their disease. Skin ulcers, amputations and abscesses are amongst the commonest.

Preoperative assessment -

Timing - diabetic patients should be placed first on the operating list. This shortens their preoperative fast. Badly controlled diabetics need to be admitted to hospital one or two days before surgery if possible to allow their treatment to be stabilised.

Hydration - Glucose in the urine (glycosuria) causes a diuresis which makes the patient dehydrated and even more susceptible to hypotension. Check for dehydration (Table 3) and start an intravenous infusion.

<table>
<thead>
<tr>
<th>Sign</th>
<th>Water loss (% body weight)</th>
<th>Thirst</th>
<th>Dry mouth</th>
</tr>
</thead>
<tbody>
<tr>
<td>Capillary refill &gt; 2 seconds *</td>
<td>&lt; 5 %</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Decreased skin turgor *</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Orthostatic hypotension *</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low intraocular pressure (soft eyes)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reduced urine output</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low CVP/JVP</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5 - 10 %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Shock</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unconscious</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt; 10 %</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Capillary refill - lift limb above level of heart, press on skin for 5 seconds, release and observe colour returning to area. Normal is < 2 seconds. Skin turgor - pinch skin on back of hand and release. Normally the fold of skin quickly falls back flat but if dehydrated stays folded or returns slowly. Orthostatic hypotension - a severe fall in BP when patient stands up causing fainting.

Medication - all medications should be continued up until surgery. Surgery causes a stress response which will change the patient's insulin requirements. Treatment will need to be adjusted according to:
- the extent of the anticipated surgery
- whether the patient is insulin dependant (IDDM) or non-insulin dependant (NIDDM)
- the quality of their blood sugar control.

In general, if the patient can be expected to eat and drink within 4 hours of surgery, then it is classified as MINOR. All surgery other than minor is classified as MAJOR. Figures 1-4 give regimes for major and minor surgery and for NIDDM and IDDM.

The aim is to keep the blood glucose level within the range 6-10 mmol/l at all times.

Special problems.

**Low Blood Sugar (hypoglycaemia)**

The main danger to diabetics is low blood sugar levels (blood glucose < 4mmol/l). Fasting, alcohol, liver failure, septicaemia and malaria can cause this. The characteristic signs and symptoms of early hypoglycaemia are tachycardia, light-headedness, sweating and pallor. If hypoglycaemia persists or gets worse then confusion, restlessness, incomprehensible speech, double vision, convulsions and coma will ensue. If untreated, permanent brain damage will occur, made worse by hypotension and hypoxia.

Anaesthetised patients may not show any of these signs. The anaesthetist must therefore monitor the blood sugar regularly if possible, and be very suspicious of any unexplained changes in the patient's condition. If in doubt, regard them as indicating hypoglycaemia and treat.

Treatment - diabetic patients learn to recognise the early signs and often carry glucose with them to take orally. If unconscious, 50ml of 50% glucose (or any glucose solution available) given intravenously and repeated as necessary is the treatment of choice. If no sugar is available, 1mg of glucagon intramuscularly will help.

**High Blood Sugar (hyperglycaemia)**

This is defined as a fasting blood sugar level > 6 mmol/l. It is a common problem found in many conditions other than diabetes eg - pancreatitis, sepsis, thiazide diuretic therapy, ether administration, glucose infusions, parenteral nutrition administration and most importantly, any cause of stress such as surgery, burns or trauma. Slightly elevated levels are thus commonly found after routine major surgery. It is usual to treat this only if the level is above 10 mmol/l.

Assess the patient, rehydrate them and delay surgery if necessary. Remember the aim is a sugar level of 6-10 mmol/l. If the sugar is below 10 mmol/l, observe and recheck it hourly throughout the operation. Should it be above 10 mmol/l, then follow the regimes in figures 1-4, according to the extent of the surgery planned.

After surgery, the insulin requirements fall as the stress response subsides. Newly diagnosed diabetics need further investigation to establish whether they will need insulin therapy, oral hypoglycaemics or indeed just diet control.

Sometimes when the blood sugar has become very high, the patient becomes comatose (diabetic coma). It is vital to correct this by adhering to the general guidelines and regimes already mentioned. Aim to reduce the sugar levels to below 10 mmol/l. When this has happened over a few days, the body uses its own fat to produce energy, and this results in high levels of waste products (ketones) in the blood and urine - this is called diabetic ketoacidosis and is a medical emergency with a significant mortality.

Diabetic ketoacidosis

This may be triggered by infections or other illnesses such as bowel perforations and myocardial infarction. The patient will be drowsy or even unconscious with fast, deep breathing due to acid in the blood. The ketones make their breath smell sweetly, like acetone. Ketones can also be detected by the use of urine and blood testing strips. Diarrhoea, vomiting, gastric dilatation (insert a nasogastric tube) or even severe abdominal pain may be present which can be misinterpreted as an acute surgical problem! As severe dehydration is usually present, surgery must be delayed until fluid resuscitation has commenced in order to avoid disastrous hypotension with induction agents. A urinary catheter will help monitor fluid balance, and an ECG and CVP line (to estimate the fluid deficit) are helpful.

The aim is to slowly return the body chemistry to normal.
Give high flow oxygen therapy.
Although the blood potassium level is usually high, the body has actually lost large amounts in the urine, and extra potassium is required intravenously. It is important to lower the blood sugar level slowly, as reducing it too fast can result in further complications such as brain oedema and convulsions. Search for infections (chest X-ray, blood and urine cultures) and treat with antibiotics. Blood gases and electrolyte measurements may also help management. figure 5 gives a regime for treatment.

Figure 1: Which Regime for my Patient?
1. Decide on the type of surgery
   Minor- patients expected to eat and drink within 4 hours of operation
   Major- all other patients
2. Then, is the patient Insulin or Non-insulin dependent?
3. Finally, are they:
   poorly controlled: delay surgery and change to soluble insulin three times daily but if surgery urgent, use Major surgery regime
   well controlled: use the appropriate regime from the Major or Minor

General Measures for all diabetics:
Measure random sugar preoperatively
4 hourly for IDDM
8 hourly for NIDDM
Test urine 8 hourly for ketones and sugar
Place first on operating list
Aim for a blood glucose of 6 - 10mmol/l

Figure 2: Minor Surgery
Non insulin Dependent Diabetics
Preoperatively- random blood sugar on admission
   < 10 mmol/l Normal medication until day of op
   > 10 mmol/l Follow as for MAJOR SURGERY
Day of operation
   Omit oral hypoglycaemics
   Blood glucose- 1 hour preop and at least once during op (hourly if op > 1 hour long) postop - 2 hourly until eating
Postoperatively
   Restart oral hypoglycaemics with first meal

Insulin dependent Diabetics
This regime only suitable for patients whose random sugar is < 10 mmol/l on admission, will only miss one meal preop & are first on the list for very minor surgery eg cystoscopy
Preoperatively
   Normal medication
Day of operation
   No breakfast, no insulin, place first on list.
   Blood glucose- 1 hour preop and at least once during op (hourly if op > 1 hour long) postop
   - 2 hourly until eating then 4 hourly
Postoperatively
   Restart normal S/C insulin regime with first meal.

Figure 3: Major surgery
All insulin dependent and non-insulin dependent who are poorly controlled (blood glucose >10mmol/l) (many NIDDM become insulin dependent during major surgery and will need managing as such. Regular glucose measurements will detect this). Normal medication until day of operation.

Day of operation

Omit oral hypoglycaemics and normal subcutaneous (S/C) insulin.

Blood glucose - check blood sugar (and potassium) 1 hour preop then 2 hourly from start of infusion at least once during operation (hourly if op > 1 hour long) at least once in recovery area 2 hourly post operatively.

Regime 1 - no infusion pump available.

Start intravenous infusion of 5 or 10% dextrose (500 ml bags) over 4 - 6 hours and add Insulin and Potassium Chloride (KCl) to each 500 ml bag as below. Change bag according to blood sugar level readings:

| Blood glucose (mmol/l) | Soluble insulin (units) to be added to bag | Blood potassium (mmol/l) | KCl (mmol) to be added to bag *
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 4</td>
<td>No insulin</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4 - 6</td>
<td>5</td>
<td>&lt;3</td>
<td>20</td>
</tr>
<tr>
<td></td>
<td></td>
<td>10</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>3 - 5</td>
<td>10</td>
</tr>
<tr>
<td>10 - 20</td>
<td>15</td>
<td>&gt; 5</td>
<td>None</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt; 20</td>
<td>20</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* If blood potassium level not available, add 10 mmol KCl.

Postoperatively
Non-insulin dependent
- stop infusion and restart oral hypoglycaemics when eating and drinking

Insulin dependent
- stop infusion when eating and drinking
  calculate the total daily dose (units) of insulin the patient was taking preoperatively
give this as S/C Soluble insulin (Actrapid), divided into 3 - 4 doses in 24 hours
this may need to be adjusted up or down until blood sugar levels stable.
  once stable restart normal regime

Remember that the patient may need additional fluids depending on surgery, blood loss etc.

Figure 4:- Major surgery - alternative regime
Regime 2 - for use with infusion pumps

The insulin and dextrose infusions are given via separate infusion pumps. This allows better control than regime 1,
but care is needed to ensure the separate lines do not become blocked, or that one infusion runs out leaving the
other infusing alone.

Insulin infusion - 50 units insulin made up to 50 ml with saline (i.e. concentration is 1 unit per ml)

<table>
<thead>
<tr>
<th>Blood glucose (mmol / l)</th>
<th>Insulin infused at (units / hour)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 5</td>
<td>0</td>
</tr>
<tr>
<td>5.1 - 10</td>
<td>1</td>
</tr>
<tr>
<td>10.1 - 15</td>
<td>2</td>
</tr>
<tr>
<td>15.1 - 20</td>
<td>3</td>
</tr>
<tr>
<td>&gt; 20</td>
<td>6 &amp; review *</td>
</tr>
</tbody>
</table>

If it is proving difficult to reduce the blood sugar level, then consider increasing the rate of insulin for each glucose level or also giving a bolus of Actrapid of 3 - 5 units.
Patients normally on higher doses of insulin will need higher rates of insulin infusion.
Dextrose infusion - 5 or 10 % dextrose infused at 100 ml per hour. Add 10 mmol KCl to each 500 ml of solution.
Post op - follow instructions as in figure 3.

Figure 5:- Treatment of Diabetic Ketoacidosis
Aims-
  - rehydration (water and salt)
  - lower blood sugar
  - correction of potassium depletion

Start an intravenous infusion of 0.9 % saline as follows-
1 litre over 30 minutes
then
1 litre over 1 hour
then
1 litre over 2 hours.
Continue 2 - 4 hourly until the blood glucose is below 15 mmol / l,
then change to 5% glucose, 1 litre 2 - 4 hrly

Up to 6 -8 litres of fluid may be required or more. Use clinical signs BP, heart rate, CVP, conscious level to judge
the amount.

Give soluble insulin (Actrapid) intramuscularly (IM) as follows-

- 20 units IM first dose then 6 units IM hourly
- measure the blood glucose hourly
- when the blood glucose is below 15 mmol/l, change to 6 units IM every 2 hours.

Once the patient has recovered and is eating/drinking, change to S/C insulin.

Potassium (K+) supplementation will be required-

- There may be a high blood potassium initially, but this will fall as the sugar level comes down.
- Measure the potassium hourly. Put 10 mmol K+ in the first litre of saline then 10 - 40 mmol in subsequent
litres of fluid, depending on the plasma level (normal 3.5 - 5.0 mmol/l).
- If potassium measurements are unavailable then put 10 mmol KCl in each litre of fluid.
- Other measures- 100 % O2. Blood gas estimation-if pH < 7.10, give 50 mmol of 8.4% bicarbonate. Usually
acidosis will correct itself slowly as the sugar comes down. Emergency surgery can start once the rehydration and
lowering of blood sugar is underway.

**Anaesthetic technique**

Intraoperative monitoring - record blood pressure and pulse every 5 minutes during the operation, and watch skin
colour and temperature. If the patient is cold and sweaty, then suspect hypoglycaemia, check the blood glucose
and treat with intravenous glucose

General anaesthesia - if gastric stasis is suspected then a rapid sequence induction should be used. A nasogastric
tube can be used to empty the stomach and allow a safer awakening. There are no contraindications to standard
anaesthetic induction or inhalational agents, but if the patient is dehydrated then hypotension will occur and should
be treated promptly with intravenous fluids. Hartmanns solution (Ringers lactate) should not be used in diabetic
patients as the lactate it contains may be converted to glucose by the liver and cause hyperglycaemia.

Sudden bradycardias should respond to atropine 0.3mg iv, repeated as necessary (maximum 2 mg). Tachycardias,
if not due to light anaesthesia or pain, may respond to gentle massage on one side of the neck over the carotid
artery. If not then consider a beta-blocker (propanolol 1mg increments: max 10mg total or labetalol 5mg
increments: max 200mg in total).

IV induction agents normally cause hypotension on injection due to vasodilatation. If a patient has a damaged
autonomic nervous system (and many diabetics do), then they cannot compensate by vasoconstricting, and the
hypotension is worsened. Reducing the dose of drug and giving it slowly helps to minimise this effect.
Regional techniques - are useful because they get over the problem of regurgitation, possible aspiration and of course difficult intubation. However, the same attention should be paid to avoiding hypotension by ensuring adequate hydration. It is a wise precaution to chart any pre-existing nerve damage before your block is inserted. With spinals and epidurals, autonomic nerve damage means the patient may not be able to keep their blood pressure in a normal range. Intervene early with ephedrine (6mg boluses) when the systolic pressure falls to 25% below normal.

Postoperative therapy regimes are also given in figures 1 - 4. It is not unusual to find that insulin requirements are reduced once the patient begins to recover from surgery.

Summary
The diabetic patient presents the anaesthetist with many challenges. Careful attention to clinical signs and rapid action to prevent even suspected hypoglycaemia peroperatively should see them safely through their surgery. The goal is to keep things as normal as possible. Regional techniques are often safer than general anaesthesia, but require the same vigilance.
ANAESTHESIA FOR THE PATIENT WITH RESPIRATORY DISEASE

Introduction
Patients with respiratory disease have an increased chance of developing complications perioperatively. Most problems are seen postoperatively and are usually secondary to shallow breathing, poor lung expansion, basal lung collapse and subsequent infection. To minimise the risk of complications these patients should be identified preoperatively and their pulmonary function optimised. This involves physiotherapy, a review of all medications and may require the help of a respiratory physician. Elective surgery is postponed until the patient is ready.

In the general surgical population thoracic and upper abdominal procedures are associated with the highest risk (10-40%) of pulmonary complications. The benefits of the proposed surgery must therefore be weighed against the risks involved.

General Considerations
General health status
The American Society of Anesthesiologists classification (1 to 5) correlates well with the risk of post-operative pulmonary complications. Poor exercise tolerance also predicts those at risk.

Smoking
Active and passive smokers have hyper-reactive airways with poor mucociliary clearance of secretions. They are at increased risk of perioperative respiratory complications, such as atelectasis or pneumonia. It takes 8 weeks abstinence for this risk to diminish.

Even abstinence for the 12 hours before anaesthesia will allow time for clearance of nicotine, a coronary vasoconstrictor, and a fall in the levels of carboxyhaemoglobin thus improving oxygen carriage in the blood.

Obesity
The normal range for BMI (Body Mass Index - defined as weight (Kg) divided by the square of the height (m) is 22-28. Over 35 is morbidly obese. Normal weight (Kg) is height (cm) minus 100 for males, or height minus 105 for females.

Obese patients may present a difficult intubation and have perioperative basal lung collapse leading to postoperative hypoxia. A history of sleep apnoea may lead to post-operative airway compromise. If practical obese patients should lose weight preoperatively, and co-existent diabetes and hypertension stabilised.

Physiotherapy
Teaching patients in the preoperative period to participate with techniques to mobilise secretions and increase lung volumes in the postoperative period will reduce pulmonary complications. Methods employed are early mobilisation, coughing, deep breathing, chest percussion and vibration together with postural drainage.

Pain Relief
Effective analgesia is important as it allows deep breathing and coughing and mobilisation. This helps prevent secretion retention and lung collapse, and reduces the incidence of postoperative pneumonia. Epidurals appear particularly good at this for abdominal and thoracic surgical procedures, although they are not available everywhere (see Epidural analgesia section).

The method of postoperative analgesia should always be discussed with the patient before surgery.

Effects of General Anaesthesia
These are relatively minor and do not persist beyond 24 hours. However, they may tip a patient with limited respiratory reserve into respiratory failure.

Manipulation of the airway (laryngoscopy and intubation) and surgical stimulation may precipitate laryngeal or bronchial spasm.

Endotracheal intubation bypasses the filtering, humidifying and warming functions of the upper airway allowing the entry of pathogens and the drying of secretions. Adequate humidification and warming of the anaesthetic gases with a Heat and Moisture Exchanger (HME) is ideal.

Volatile anaesthetic agents depress the respiratory response to hypoxia and hypercapnia, and the ability to clear secretions is reduced. Functional residual capacity (FRC) decreases and pulmonary shunt increases; these are unfavourable changes leading to hypoxia and occur especially in lithotomy and head-down positions, and in the obese.

Intermittent positive pressure ventilation causes an imbalance in ventilation and perfusion matching in the lung, and necessitates an increase in the inspired oxygen concentration. Excessive fluid therapy can result in pulmonary oedema in patients with cardiac failure.

Neuromuscular blockade is reversed before extubation. In the recovery room residual effects of anaesthesia depress upper airway muscular tone, and airway obstruction may occur.
**Anaesthetic Drugs**

The intravenous induction agents thiopentone, propofol and etomidate produce an initial transient apnoea. Ketamine preserves respiratory drive and is better at maintaining the airway, although secretions increase. Thiopentone increases airway reactivity.

Volatile anaesthetics depress respiratory drive in decreasing order as follows:

- Enflurane > Desflurane > Isoflurane > Sevoflurane > Halothane.

Ether however stimulates respiratory drive and increases minute ventilation. It is, however, irritant to the airway, stimulates saliva production and may induce coughing.

Atracurium and tubocurare release histamine and may result in bronchospasm. They are best avoided in asthma.

Opioid drugs and benzodiazepines depress respiratory drive and response to hypoxia and hypercapnia.

Morphine may result in histamine release and occasionally bronchospasm. Non-steroidal anti-inflammatory drugs (NSAIDS) may exacerbate asthma. Pethidine is a useful alternative analgesic for asthmatics.

**Effects of Surgery**

To immobilise upper abdominal and thoracic incisions and limit pain, patients splint these areas postoperatively with their intercostal and diaphragmatic muscles. This limits their ability to take deep breaths and increases the risk of postoperative pulmonary complications. Surgery on the limbs, lower abdomen or body surface surgery has less effect.

A laparotomy may remove fluid or masses that cause diaphragmatic splinting and respiratory difficulty. However, gas (especially nitrous oxide) and fluid may accumulate within the bowel and peritoneal cavity exacerbating post-operative distension and splinting.

Surgery lasting more than 3 hours is associated with a higher risk of pulmonary complications. Postoperatively, return of lung function to normal may take one to two weeks.
A MOBILE ANAESTHESIA SERVICE

Some of the more invasive procedures on the ward are painful and frightening for patients. This is particularly true in children who may be too young for local anaesthetic techniques alone. These patients are managed in a variety of ways in different hospitals but many require more analgesia than can be provided with parenteral morphine. Some hospitals take all such patients to theatre which has many advantages in terms of equipment and resources, others take equipment to a dedicated area of the routine ward.

Effective anaesthesia / sedation for procedures requires a combination of drugs that affect vital reflexes. To make the procedure as safe as possible, the patient needs to be directly supervised by someone with anaesthesia training and skills. This paper discusses our approach in Tansen, where we have developed a system which allows us to provide sufficient analgesia to perform small “ward-procedures” effectively.

Indications
These include painful examinations, such as
- Lumbar punctures in children
- Dressing changes
- Manipulations
- Therapeutic as well as diagnostic procedures.

Preparation
The ward nurses prepares equipment normally kept on the ward. Extra equipment is brought by the anaesthetic nurse at the time of the procedure. Patient has to be fasted: 4 hours breast milk, 6 hours food, 2 hours clear fluid. Usually the procedure is performed in the morning, so patients can have their breakfast afterwards

Procedure
At the arranged time the anaesthetist, anaesthesia nurse, patient and ward nurse are ready. After inducing the patient, the procedure is performed. The anaesthesia nurse stays with the patient until they are completely awake. Monitoring is by clinical observation, but a pulse oximeter is used whenever possible. The drugs most commonly used are 0.2mg/kg diazepam iv + 0.5-1mg/kg ketamine iv.

Results
Over a 6 month period (April to September 2001) we anaesthetised 250 patients on the wards. The cost per procedure for the anaesthetic (including salaries, drugs etc.) was calculated with $1, which made the service self-sustaining. No complications were encountered.

Discussion
Ketamine, covered with diazepam (to prevent emergence reaction), can give adequate analgesia for painful procedures where local anaesthesia alone is inadequate or unsuitable. Essential anaesthetic precautions must be taken with an experienced anaesthesia practitioner. With careful attention to safety, the complication rate is very low (e.g. comparing with high doses of opiates), the analgesic effect is significantly better and the side effects reduced.

There are also potential savings in theatre time.

Ward nurse
- airway and breathing - oxygen, suction, self-inflating bag, anaesthetic masks,
- oro-pharyngeal airways, laryngoscope, endotracheal tubes, intubating stylet, tape
- intravenous - canulas and fluids, needles, syringes
- drugs - adrenaline, atropine, lignocaine, diazepam, diclofenac, sterile water, sodium chloride
- various - ampoule-cutter, tourniquet, syringes, sterile gloves
- tilting trolley - if possible

Anaesthetic nurse
- drugs - ketamine, diazepam, suxamethonium (needs to be taken out of the fridge), atropine, ephedrine
A PRACTICAL APPROACH TO EMERGENCY EYE ANAESTHESIA

Introduction

Anaesthesia for emergency eye surgery can present special problems to the anaesthetist. An understanding of some basic principles and techniques of eye anaesthesia have been discussed in previous issues of Update (Anaesthesia for Ophthalmic Surgery - Part 1: Regional Techniques, Update in Anaesthesia 1996;6:3 and Anaesthesia for Ophthalmic Surgery - Part 2: General Anaesthesia, Update in Anaesthesia 1998;8:5).

This article discusses the specific problems of emergency anaesthesia for eye surgery. We try and answer the common questions concerning these patients and provide a practical guide.

Indications for emergency eye surgery

An emergency is defined as an event that has to be dealt with immediately, usually within the first hour after presentation. The commonest eye emergencies that fall into this category are chemical burns of the eye and retinal artery occlusion. Neither of these requires surgery as part of the initial management. The majority of cases presenting as emergencies can therefore be defined as urgent cases.

Trauma is by far the commonest indication for urgent surgery. Traumatic injuries can be blunt or penetrating ("open eye"). The incidence is highest in young adult males and children. Trauma is often associated with industrial or motor vehicle accidents. Eye protection in the workplace and car safety belts have lowered the incidence of eye trauma in many countries. Eye trauma is usually confined to one eye. Some patients may present with trauma to both eyes or with multiple injuries.

Non-traumatic surgical "emergencies" include spontaneous retinal detachment, infections, and complications of previous surgery. One of the factors which determines the degree of urgency for retinal detachment surgery is the condition of the macula. The risk of a detachment progressing and resulting in loss of the macula increases the sense of urgency. There is usually enough time however to allow for fasting prior to surgery.

Timing of surgery

Ideally all patients should be fasted before undergoing general anaesthesia to minimise the risk of aspiration and subsequent lung injury. This obviously has to be weighed against the risk to the eye that delaying surgery may cause. It is essential to liaise closely with the surgeon to establish the degree of urgency. Most cases involving blunt trauma can usually be delayed to allow for patient fasting.

Penetrating injuries may need to be dealt with more urgently due to the risk of infection and endophthalmitis. If the patient has an open eye injury there is also the risk of vitreous loss and retinal detachment. Even with open eye injuries many ophthalmic surgeons are willing to delay surgery until a patient is adequately fasted prior to anaesthesia.

This is especially the case where there is severe damage to the eye and surgery is not going to improve sight. This group of patients are usually admitted for bed rest and have an eye shield covering the injured eye until they are ready for primary closure of their eye wounds. Open eye injuries in which the eye is still largely intact and the visual prognosis is good need to be dealt with more urgently. Decision making needs to be made on a case by case basis.

The degree of urgency will depend on the size of the laceration and commensurate risk of loss of ocular contents, how dirty the wound is and the risk of infection.

A fast of six hours is normally suggested in the uncomplicated patient. It is now common practice to allow patients to drink clear fluids (water, non-fizzy fruit drinks) up to two to four hours prior to the time of surgery. In patients who have had trauma or received opioids, it can take up to 24 hours for gastric emptying to take place. The most important time interval is that between the last meal and the time of the injury. If trauma occurs soon after a large meal the patient may still have a full stomach after the standard six hour fast. Alcohol also delays gastric emptying. If surgery is necessary in a patient with a full stomach then a rapid sequence induction technique should be used (see below).

How long patients should be fasted for prior to surgery with a local anaesthetic block is controversial. We feel that in the patient undergoing emergency eye anaesthesia the above principles regarding fasting should be used irrespective of the anaesthetic technique chosen.

Does the patient have other medical problems?

Eye trauma requiring surgery may be associated with other injuries that may or may not require surgery. In the multiply injured patient normal trauma principles must always be applied. Life-threatening problems should be dealt with before sight-threatening problems. The principles of managing the patient with major trauma have been discussed in Anaesthesia for Ophthalmic Surgery - Part 1: Regional Techniques, Update in Anaesthesia 1996;6:3.
Patients with other disease processes such as diabetes or ischaemic heart disease should have these optimised prior to surgery if time allows.

**Choice of a local or general anaesthetic technique**

The choice of technique will depend on patient factors as well as local facilities and surgeon preferences. In many countries extra-ocular, anterior segment and vitreo-retinal eye surgery is routinely performed using local anaesthetic techniques. However there are many practical reasons why a general anaesthetic is often preferable for emergency cases. Firstly, the patient must be able to lie flat, still and protect his or her own airway safely for the duration of the procedure. Thus, children, uncooperative or intoxicated patients are usually better candidates for a general anaesthetic. An uncooperative patient with an open eye is extremely difficult to manage. Spread of local anaesthetic agents is poor in patients with eye and orbital infections. Some procedures such as scleral banding (scleral buckling) for retinal detachment can be extremely uncomfortable even with a good local anaesthetic block. In our experience younger adults tend to tolerate surgery with a local anaesthetic technique poorly compared with elderly patients.

In open eye injuries local anaesthetic techniques are usually avoided. Injection of local anaesthetic using peribulbar and retrobulbar techniques is associated with an increase in intra-ocular pressure which may lead to vitreous loss. Oculoclosure after the block is also not an option if the patient has an open eye injury. In some patients it may be possible to operate on small open eye injuries using topical anaesthesia, sub-tenon blocks or a careful peribulbar or retrobulbar block.

**Is sedation an option?**

Sedation should be used cautiously. Oversedation can easily turn a cooperative patient into a difficult to manage patient due to airway problems and patient confusion. Sedation should not be used as an alternative to a general anaesthetic in a patient with a full stomach. If a patient develops pain during surgery using a local anaesthetic technique the patient requires analgesia and not sedation. The surgeon should supplement the block using local anaesthesia or small doses of intravenous analgesia should be given.

If sedation is to be used then small doses of a short acting agent such as midazolam should be given. Diazepam in small doses may also be an option. Propofol in small 10mg increment doses can also be used especially prior to performing a local anaesthetic eye block. Some anaesthetists use small doses of alfentanil or fentanyl. The key to good sedation is to maintain verbal contact with the patient.

Careful surgical draping is also important. Patients become claustrophobic if their faces are draped. Use of a bar to hold up the drapes can allow a tent to be made to allow better ventilation (figure 1). Oxygen should be given to the patient, especially if sedation is to be used. Patients may find a face mask or nasal oxygen cannulae uncomfortable. Oxygen can be insufflated under the drapes using a breathing circuit. This also improves air circulation under the drapes.

Many of the problems associated with local techniques can be avoided with a clear explanation of the procedure to the patient prior to commencing surgery, having a comfortable operating table, and somebody to hold the patient’s hand throughout. Allowing patients to empty their bladders prior to surgery also helps.

**Choice of drugs for general anaesthesia**

The choice of intravenous induction agent will depend on local availability and user familiarity. Most intravenous induction agents reduce intra-ocular pressure therefore preventing further damage to the injured eye. Ketamine possibly raises intra-ocular pressure although the literature is conflicting. Most textbooks state that it should be avoided in open eye injuries. If it is to be used it is best to use it in combination with small doses of a benzodiazepine (midazolam, diazepam) to blunt its excitatory effects. The majority of problems with ketamine and intra-ocular pressure seem to occur when it used as a sole agent in a patient with an unprotected airway breathing spontaneously. Ideally ketamine should be used with a muscle relaxant and controlled ventilation if intra-ocular pressure control is important.

All the non-depolarising muscle relaxants can be used without adverse effects on the eye so choice will depend on availability. Suxamethonium (scoline) increases intra-ocular pressure. The exact mechanism is unclear but it is not thought to be solely due to contraction of the extra-ocular muscles. Suxamethonium also causes an increase in the intra-ocular blood volume and this may contribute to the rise in intra-ocular pressure. The rise in intra-ocular pressure occurs after one to two minutes and wanes after six to ten minutes. The extent of the rise in intra-ocular pressure will depend on the other drugs used and the response to laryngoscopy and intubation. Its use in penetrating eye injury anaesthesia is controversial. The majority of eye surgeons prefer if it is not used. Adequate fasting prior to surgery will allow suxamethonium to be avoided for the majority of urgent cases. This obviously presents a dilemma in the patient with a full stomach as suxamethonium is used as part of a ‘rapid sequence induction’ to enable an airway to be secured quickly. In this situation the relative risks need to be weighed, i.e. prevention of aspiration (potentially life threatening) versus ocular damage (potentially sight threatening).
Suxamethonium avoiding techniques include the use of large doses of vecuronium or pancuronium to speed up its onset of action as part of a modified rapid sequence induction technique. The non-depolarising neuromuscular blocker rocuronium has a rapid onset of action with a duration of 30 to 40 minutes. It can be used for a rapid sequence induction technique but can only be recommended to those who have gained experience in its use and for patients in whom airway problems are unlikely to occur. On balance there are no case reports of ocular damage with suxamethonium use, and no good evidence that suxamethonium-avoiding techniques are any better or safer.

**Airway management and mode of ventilation**

It is considered good practice to intubate and ventilate the patient to ensure a secure airway (the surgical field is in close proximity) and to facilitate mild hypocarbia (this reduces intra-ocular pressure). The laryngeal mask airway is a popular choice for airway management for elective eye surgery in the UK. Laryngeal mask insertion avoids the pressor response to laryngoscopy and intubation causing raised intra-ocular pressure. The laryngeal mask does not protect against aspiration of gastric contents. Its use in emergency anaesthesia is therefore limited.

**Analgesia and control of nausea and vomiting**

It is possible to manage pain in the majority of patients after eye surgery with oral analgesia. Avoiding opioids if possible helps prevent nausea and vomiting. Regular doses of paracetamol (acetaminophen) and a non-steroidal anti-inflammatory drug (ibuprofen, diclofenac, ketoprofen) should be prescribed. Codeine phosphate can also be added. These drugs are best accepted by children if given as an elixir (syrup). Some analgesic drugs are listed in the table below.

<table>
<thead>
<tr>
<th>Analgesic Drugs</th>
<th>Drug</th>
<th>Dose</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paracetamol</td>
<td>(Acetaminophen)</td>
<td>Children: 90 mg/kg total per 24 hours orally or rectally in 4-6 divided doses</td>
<td>Avoid if liver dysfunction. Decrease dose to total of 60 mg/kg per 24 hours if treatment for more than 48 hours.</td>
</tr>
<tr>
<td></td>
<td>adults: 1g orally or rectally. 4g total per 24 hours</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ibuprofen</td>
<td>Children: 10mg/kg orally. 4 doses maximum in 24 hours.</td>
<td></td>
<td>Ibuprofen has the lowest side effect profile of the non-steroidal anti-inflammatory drugs. Avoid in renal and peptic ulcer disease. Use with care in asthma. Not in children &lt;7kg.</td>
</tr>
<tr>
<td></td>
<td>Adults: 400 mg orally. 4 doses maximum in 24 hours.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diclofenac</td>
<td>Children: 1mg/kg orally or rectally. 3 doses in 24 hours.</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Adults: 150 mg total by any route in 24 hours</td>
<td></td>
<td>Cautions as for Ibuprofen.</td>
</tr>
<tr>
<td>Codeine Phosphate</td>
<td>0.5 mg/kg orally 6 hourly</td>
<td>Use with care when co-administered with other opioids</td>
<td></td>
</tr>
</tbody>
</table>
In patients having surgery with general anaesthesia it is a good idea to ask the surgeon to perform a local anaesthetic block before waking up the patient. If stronger analgesia is required this is best given as small intravenous doses of morphine or pethidine.

Nausea and vomiting after emergency eye anaesthesia can be a major problem in some patients. Anti-emetic prophylaxis may help prevent this. Some patients may benefit from a regular anti-emetic in the post-operative period.

There is a vast number of anti-emetic drugs available. Most have a limited efficacy. Using a combination of small doses of anti-emetic drugs from different pharmacological classes may enhance efficacy and reduce side effects.

Some anti-emetic drugs are listed in the table below.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Droperidol</td>
<td>0.5 to 1 mg in adults. Up to 3 times a day</td>
<td>Cheap and effective but causes drowsiness, sedation, anxiety and restlessness. Risk of extrapyramidal effects.</td>
</tr>
<tr>
<td>Cyclizine</td>
<td>Children 1mg/kg iv</td>
<td>Anti histamine and anti-cholinergic effect.</td>
</tr>
<tr>
<td></td>
<td>Adults 50 mg iv</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Up to 3 times a day</td>
<td></td>
</tr>
<tr>
<td>Ondansetron</td>
<td>Children 0.1 mg/kg iv</td>
<td>Expensive but effective with low side effect profile.</td>
</tr>
<tr>
<td></td>
<td>Adults 4 mg iv</td>
<td></td>
</tr>
<tr>
<td></td>
<td>3-4 doses per 24 hours</td>
<td></td>
</tr>
</tbody>
</table>

A practical approach to emergency eye anaesthesia

1. Assess the indication for emergency anaesthesia in discussion with the surgeon. Can surgery be deferred until normal working hours and to allow adequate fasting?
2. Carry out a full preoperative assessment including a history and examination.
3. Are there any medical/trauma issues that need addressing first?
4. Decide on choice of anaesthetic technique. Provide the patient with a full explanation. Tell the patient what to expect if a local anaesthetic technique is to be used.
5. If a general anaesthesia is chosen decide if the patient has a full stomach and is at risk of aspiration.
6. If the patient has a full stomach a rapid sequence induction technique should be used. They should be preoxygenated with 100% oxygen. Pressure on the affected eye from the mask must be avoided. The patient should then be induced with an intravenous anaesthetic agent (e.g. thiopentone 4-7mg/kg) and a rapid onset muscle relaxant (suxamethonium 1-1.5mg/kg is currently the only realistic option). While the patient is being induced cricoid pressure should be applied by an assistant (Sellick's manoeuvre) thus occluding the oesophagus behind. The patient’s trachea should be intubated after which the cricoid pressure can be removed. Note that the endotracheal tube tie should not be tight around the neck as this impedes venous drainage and raises intra-ocular pressure.
7. Choice of maintenance depends on local availability e.g. 40% O2, 60% N2O and an inhalational agent. Note that all inhalational agents reduce intra-ocular pressure.

8. Control ventilation during the procedure aiming for low to normal end-tidal carbon dioxide. This may require the use of a longer acting muscle relaxant (e.g. vecuronium 0.1mg/kg). A slight head up tilt helps reduce intra-ocular pressure.

9. At the end of the procedure the patient should be extubated on their side and once airway protective reflexes have returned. In patients not deemed at risk of aspiration extubation with the patient deep and breathing spontaneously may prevent coughing. Severe coughing and straining needs to be avoided as this increases the risk of ocular haemorrhage.

10. If the patient does not have a full stomach and is not deemed at risk of aspiration, general anaesthesia should proceed as for an elective patient. Pre-oxygenate the patient for safety and induce with an intravenous agent. Give a long acting muscle once ability to hand ventilate is established. Laryngoscopy should be performed gently. Consider spraying the vocal cords with lignocaine to minimise the pressor response to intubation. This may also decrease the risk of coughing on intubation. Intubate, ventilate and maintain anaesthesia as above.

11. Post-operatively nausea, vomiting and pain should be kept to a minimum as they can cause rises in intra-ocular pressure. Prescribe regular oral analgesia and an anti-emetic. Some patients may need stronger analgesia early after surgery. Titrate small doses of intravenous opioid (morphine, pethidine) to control pain.
ANAESTHESIA FOR THE ELDERLY PATIENT

Introduction
Increasing numbers of elderly patients are presenting for surgery due to longer life expectancy. The incidence of peri-operative complications is much higher in these patients due to reduced functional reserve and a high incidence of co-morbidity, but these complications can be minimised by careful preoperative assessment, a meticulous anaesthetic technique and good postoperative care.

Age-Related Physiological Changes
Ageing is a process where progressive cell loss occurs, at a variable rate, in individual patients and their organ systems. The concept of “functional reserve” is derived from the difference between the basal level of organ function at rest and the maximum level of organ function that can be achieved in response to increased demand, for example during exercise or in response to surgical stress. Functional reserve is often reduced in elderly patients, and is thought to be a major factor in the increased morbidity and mortality of the elderly population. However, decreased functional reserve may be difficult to detect. Some patients are limited by lack of mobility and as a result do not exert themselves as much. These patients rarely admit to breathlessness or angina, yet they may have significant underlying and undetected ischaemic cardiac disease.

Alterations In Organ Function
Almost all age-related changes in organ systems are relevant to the anaesthetist. However, reduction in cardiovascular, pulmonary, renal and central nervous system function may be the most important determinants of outcome from surgical procedures under general or regional anaesthesia.

Cardiovascular system
Ischaemic heart disease is common in affluent societies. Smoking, hypercholesterolaemia, hypertension, type 2 diabetes mellitus and obesity all contribute to the development of atherosclerosis. The result is a less compliant arterial tree, increased systemic vascular resistance and systemic hypertension. The net effects on the heart are concentric left ventricular hypertrophy, reduced ventricular compliance and contractility, and eventually reduced cardiac output.

In contrast, valvular heart disease secondary to rheumatic fever is more commonly seen in developing countries. Over 50% of patients will have mitral valve disease. Aortic lesions are less common.

The reduced cardiac output in heart disease compromises blood flow to the kidneys and brain. Autoregulation of blood flow to these organs is impaired in the elderly, and therefore both the kidneys and brain are prone to peri-operative ischaemia.

The physiological response to cardiovascular stressors (such as hypovolaemia) may be blunted due to reduced baroreceptor sensitivity and autonomic function. This lack of compensation may be significant if the patient is taking medication such as beta-blockers or ACE inhibitors. A normal response to exercise in young patients is an increased heart rate and ejection fraction. This response is blunted in elderly patients, due to decreased reactivity of β receptors, and as a result the ejection fraction may even fall. Maximum cardiac output and hence functional cardiac reserve decreases as age increases.

Atrial fibrillation (AF) in the elderly population is common, probably due to a progressive loss of atrial pacemaker cells with ageing. A 70 year old adult has only 10% of the atrial pacemaker cells that an adolescent has. The fast ventricular rate in AF leads to poor diastolic filling and reduced cardiac output: both are poorly tolerated in an elderly patient. Preoperatively, a patient in AF should ideally be cardioverted, but failing this the ventricular rate should be controlled to <100/minute.

Respiratory system
Pulmonary elasticity, lung and chest wall compliance, total lung capacity (TLC), forced vital capacity (FVC), forced expiratory volume in one second (FEV1), vital capacity (VC) and inspiratory reserve volume (IRV) are all reduced, with an increase in the residual volume. Although functional residual capacity (FRC) is unchanged, closing capacity rises progressively with age, and may become greater than the FRC - this occurs in the supine position at 44 years of age and in the upright position at 66 years. The end result of these changes is airways collapse, VQ mismatch and hypoxaemia, even during tidal volume breaths. The small airways and alveoli therefore have to be reopened at each inspiration, leading to increased work of breathing and possible difficulties weaning from ventilation. The efficiency of gas exchange is reduced, and as a result PaO2 decreases with age (PaO2 = 13.3-age/30 kPa, or PaO2 = 100-age/4mmHg) although PaCO2 remains constant.

Atelectasis, pulmonary embolism and chest infections are all more common in elderly patients, particularly following abdominal or thoracic surgery. Ineffective mucociliary activity exacerbated by smoking increases the risk of complications. Early mobilisation and good analgesia following abdominal surgery help reduce lung atelectasis and collapse.
Renal system
Glomerular filtration is reduced. Muscle bulk decreases with age resulting in reduced creatinine production, hence even a modest rise in serum creatinine may represent significant renal impairment. Tubular function is also impaired, with reduced renal concentrating ability and reduced free water clearance. Clearance of renally excreted drugs is reduced, and fluid balance is more critical as responses to both fluid loading and dehydration are impaired. Renal function may deteriorate rapidly in hypovolaemic patients, particularly those taking NSAIDs (non-steroidal anti-inflammatory drugs) or ACE inhibitors such as captopril. Close monitoring of hourly urine output after major surgery should be routine.

Nervous system
An age-related decline in central nervous system (CNS) function is common, the causes of which include cerebrovascular disease, changes in hormone levels, neuronal damage induced by oxidative stress as well as a generalised progressive loss of cells. As a result, confusion is more common, both pre and post-operatively. Cognitive impairment increases with ageing, and dementia may affect up to 20% of patients over 80 years of age. However, it is important that dementia is only diagnosed after formal testing, ideally by specialists in geriatric psychology. Blindness affects nearly 30% of the elderly, largely due to cataracts and glaucoma, and may make understanding written material such as consent forms and visual analogue pain scales very difficult. Deafness is more common, and may be severe in about 35% of elderly patients. Autonomic dysfunction is also more prevalent in the elderly population, and may result in labile blood pressure and arrhythmias perioperatively. The baroreceptor reflex may be attenuated, leading to postural hypotension and a drop in blood pressure during anaesthesia, particularly during induction in a relatively hypovolaemic patient. Impaired temperature regulation and delayed gastric emptying may also occur, the latter predisposing the patient to aspiration. A rapid sequence induction should therefore be performed in such cases.

Endocrine
The incidence of diabetes is increased in the elderly, and may be seen in up to 25% of patients aged over 80 years. Diabetics frequently have cardiovascular, renal, neurological and visual impairment, and require control of blood glucose levels during the perioperative period. (See Update in Anaesthesia issue 10)

Pharmacology
Pharmacokinetics may be altered, with reduced hepatic and renal blood flow and a reduction in total body water. Plasma proteins are often reduced, resulting in reduced protein binding of drugs and metabolites, thereby increasing free drug levels and possible toxic effects. Pharmacodynamics may also be altered, with increased sensitivity to many agents, especially CNS depressants. Minimum alveolar concentration (MAC) decreases steadily with age by 4-5% per decade after 40 years – for example the MAC of isoflurane is approximately 0.92 at 80 years of age.
It may be difficult to ascertain exactly which medications are being taken, especially when patients are admitted as an emergency. Patients may be confused as to what drug/drugs they are taking, compliance may be poor, or medication may have been inadvertently stopped. It may be necessary to confirm exact details of current medication with a patient’s relatives or family doctor. Long-term medication should usually be continued throughout the hospital stay.

Nutrition
Malnutrition is common in the elderly, and is associated with increased morbidity and mortality. Trials of nutritional supplementation reduce the length of hospital stay and postoperative complications. Consider oral protein supplementation in those with significant malnutrition.

Musculoskeletal
Degenerative diseases of all types affect the elderly, and arthritis is almost universal. This may limit exercise tolerance and makes it difficult to assess fitness. Osteoporosis and ligament laxity makes epidurals and spinals technically difficult; in addition, the patient is prone to fractures or dislocation of joints (including the cervical spine) while anaesthetised. Care should be taken with patient movement and intra-operative positioning. Vulnerable pressure points should be well padded.

PREOPERATIVE PREPARATION
Assessment
- A full history and thorough clinical assessment is required - significant cardiac, respiratory and renal disease may not have been previously detected. An ECG is required for all patients. A chest X-ray should be arranged for patients with known malignancy or possible tuberculosis, and for anyone with symptomatic cardiovascular or respiratory disease who has not had a recent chest X-ray. Note the level of cognitive
function and the patient’s social circumstances: these may determine both the perioperative prognosis and plans for the patient’s rehabilitation postoperatively.

- In patients who have sustained a fracture, actively look for an underlying medical cause for a fall, such as arrhythmias, myocardial infarction, transient ischaemic attack (TIA), cerebral vascular event (CVE), pulmonary embolus, gastrointestinal bleed.

- Assessment of exercise tolerance and functional ability is important. The baseline functioning of the patient should be well documented. If a decreased functional reserve is detected, a high-care or intensive care facility may be appropriate post-operatively.

- A full explanation of the perioperative period should be given (details such as catheters, nasogastic tubes, CVP lines are important so the patient is expecting these when awakening). The patient should be consented for anaesthesia. If the patient will be on a different ward postoperatively, a preoperative visit may reduce confusion after the operation.

- The American Society of Anaesthesiologists (ASA) score should be recorded as it remains a good predictor of outcome in the elderly.

Resuscitation/optimisation pre-operatively

Dehydration is common (note large fluid losses are associated with routine bowel preparation, and it is common to lose 50 - 1000mls of blood with a femoral neck fracture, especially with an extracapsular or trochanteric fracture.). Consider prescribing preoperative fluids if not already done.

One issue that is currently being debated in the anaesthetic press is whether patients, and especially elderly patients with ischaemic heart disease, may benefit from preoptimisation. This describes the enhancement of oxygen delivery to the tissues during the perioperative period, by using fluid therapy, oxygen and possibly inotropic agents.

One high profile study in the BMJ showed a significant reduction in mortality following major surgery by using fluid and inotropic therapy along with invasive haemodynamic monitoring, but as yet this has not become routine practice in the UK.

Consider day case surgery

The advantages of this include less confusion, earlier mobilisation and less nosocomial infections. However, day case surgery does need meticulous planning and preoperative assessment, including a detailed social appraisal as to the level of home support and care available.

Decision to operate.

Extensive surgery may be futile in certain patients. Sometimes the best decision is not to operate and this should be made at consultant level, ideally in consultation with the patient and other members of the family.

PERIOPERATIVE CARE

In general the full range of anaesthetic drugs and techniques used for young, fit adults may be used in elderly patients, within the limitations of their physiology. Modification of the techniques, and particularly drug doses, may be required.

Induction of anaesthesia

Arm-brain circulation time is increased, and induction agent dose requirements are drastically reduced. Titrate drugs slowly against effect, and inject into a running intravenous infusion. Thiopentone or propofol are both useful but should be given slowly to avoid overdose. An induction dose of propofol may result in hypotension and require a vasopressor. Avoid ketamine in the presence of cardiac disease as the tachycardia and hypertension that may result can increase myocardial oxygen consumption and precipitate ischaemia. However, bear in mind that ketamine’s hallucinogenic effects are not as marked in the elderly, and that it remains a very safe and effective analgesic, anaesthetic and sedative.

Maintenance of anaesthesia

Maintenance of anaesthesia with inhalational agents is a suitable technique for elderly patients, as the depth of anaesthesia can be rapidly changed and inhalational agents are minimally metabolised. Isoflurane is maybe the most suitable, as it is relatively cardiovascularly stable, has a short onset and offset time and only 0.2% of an administered dose is metabolised. Halothane has the advantage of being non-irritant to the upper airway and respiratory tract, although it sensitises the myocardium to catecholamines and so may predispose to tachyarrhythmias. Ether has been used successfully for many years, and in elderly patients is best given in low concentrations with supported ventilation. This allows the patient to wake up more quickly than prolonged deep ether anaesthesia.

Temperature

Maintenance of body temperature pre-, intra- and postoperatively is essential. Elderly patients have a reduced basal metabolic rate (BMR) and are susceptible to heat loss as a result of impaired thermoregulation. Shivering
may increase oxygen demand significantly and so should be avoided whenever possible. Conservation of heat by wrapping a patient up (including the head if possible), using fluid warmers and active warm air systems if available, and by operating in a warm ambient environment all help maintain body temperature and aid recovery.

**Fluid management**

Careful peri-operative fluid balance is mandatory in the elderly. Always consider measuring the CVP with large fluid shifts. Patients are more often underfilled than overloaded, although care should be taken to avoid fluid overload: excess fluids in an elderly patient, especially in the presence of renal failure, can cause pulmonary oedema. Conversely, dehydration, which can be difficult to assess in the elderly, can precipitate renal failure. Regular review of fluid therapy is essential after major surgery.

**Pressure areas**

Most pressure sores develop within the first 24 hours after surgery, and are more common in patients who have undergone long procedures, and those who have been exposed to periods of hypotension and poor tissue perfusion. Pressure sores should be avoided as they prolong hospital stay, delay rehabilitation and may cause sepsis. Suitable measures to prevent sores should be taken in both the operating theatre and recovery areas.

**General or regional anaesthesia?**

Regional anaesthesia may have some advantages over general anaesthesia, including less thromboembolic events, confusion and respiratory problems post-operatively. Limb and plexus anaesthesia are ideal for peripheral surgery. Hernias and cataracts are widely performed under local anaesthesia.

Hypotension is more commonly seen in elderly patients undergoing spinal/epidural anaesthesia due to impaired autonomic function and reduced compliance of the arterial tree. In patients with severe cardiovascular disease who require tight control of their blood pressure, general anaesthesia may be better. The Cochrane Review of anaesthesia for hip fracture surgery looked at 17 trials (involving a total of over 2800 patients) comparing regional and general anaesthesia. It concluded that regional anaesthesia may reduce mortality at one month, but that regional and general anaesthesia appear to produce comparable results for longer term mortality.

**POSTOPERATIVE CARE**

**Oxygen therapy**

It is good practice to prescribe post-operative oxygen therapy for all elderly patients, and especially following abdominal or thoracic surgery, in the presence of cardiovascular or respiratory disease, in situations where there has been significant blood loss, or when opioid analgesia has been prescribed. Nasal cannulae are often better tolerated than facemasks.

**High dependency care**

If high dependency care or intensive care facilities are available, these may improve the long-term outcome of elderly patients, especially those undergoing urgent or emergency surgery.

**Analgesia**

Consider prescribing a regular simple analgesic such as paracetamol, and use NSAID’s with caution; the complications of NSAIDs, including renal impairment and peptic ulceration, are more prevalent in older patients. Intramuscular and subcutaneous opioids may be unreliably absorbed due to variable tissue perfusion, and an elderly confused patient may have difficulty using a PCA. Regional techniques or an iv opioid infusion (with appropriate close supervision) may be the most appropriate method of pain relief. Involve an acute pain team whenever possible and consider using pain assessment charts: these should include regular pain and sedation scoring, using recognised non-verbal scoring systems if possible. The use of such pain assessment charts has been shown to improve pain management and to reduce the complications related to post-operative analgesia.

**Fluid management**

Meticulous fluid management continues to be extremely important during the post-operative phase. Fluid balance charts should be utilised and carefully interpreted: failure to do so has been shown to be a major contributing factor in post-operative morbidity and mortality.

**Other considerations**

- Frequent and regular review of the patient should be routine.
- Early and frequent physiotherapy and mobilisation facilitate post-operative recovery and have been shown to reduce hospital stay significantly.
- Consider deep vein thrombosis (DVT) prophylaxis: elderly patients are a high-risk group, especially those with a fractured neck of femur or those who have been bed bound for some days.
- Regular review looking for postoperative complications. Common complications include infection (especially wound, chest, urine), DVT and pulmonary embolus. Confusion may also be seen, and may be due to sepsis,
dehydration, overhydration, abnormal urea and electrolyte levels, hypoxia, alcohol/drug withdrawal or pre-existing cognitive impairment/dementia.

- Parenteral or enteral nutrition should be continued from the pre-operative period, or instigated early after surgery to facilitate healing and aid recovery.
- Rehabilitation using a multidisciplinary team is strongly recommended.
ANAESTHESIA FOR THE PATIENT REQUIRING EMERGENCY ABDOMINAL SURGERY

Introduction
The principles of anaesthesia for the patient requiring emergency abdominal surgery are common to adults and children, and to the patient and their anaesthetist wherever they are, and whatever resources are available. Within this framework, the article will address the importance of attention to:

- Airway
- Breathing
- Circulation
- Drugs
- Equipment
- Fluids and electrolytes

The major part of the article is about general anaesthesia, with some comments on regional anaesthesia, which may be the only option on some occasions.

The Anaesthetist and the Environment
The anaesthetist has to bear in mind a number of things when preparing to anaesthetise a patient for emergency abdominal surgery. This not only includes the patient’s condition, and the nature of the surgery, but also the anaesthetist’s own knowledge and experience, the anaesthetic equipment, and the consumables and drugs which are available.

The anaesthetist must take into account issues such as the knowledge and experience of the surgeon, the availability of an anaesthetic assistant, and the reliability of services such as oxygen, suction and power. For emergencies, particularly, there is often no second chance should a crisis occur, and if fall-back plans have not been made before starting the anaesthetic.

Pre-Aanaesthetic Check
The operating theatre needs to be always ready for an emergency procedure, so the anaesthetist does not have to waste time cleaning up and finding things used in the previous case. A systematic approach is necessary. An example is to check the following (Table 1).
Fresh gas supply - is it air, or oxygen. If it is oxygen, is it supplied by an oxygen concentrator, a cylinder, or from a wall outlet? What reserves are there in theatre or in the bulk supply?

Gas delivery system - is it draw-over, demand flow, or continuous flow?

Anaesthetic delivery - will nitrous oxide be used? Is the main agent ether, halothane, enflurane, isoflurane, sevoflurane? Is the vaporiser full, and does it work? Is it draw-over or plenum? Is there extra agent available?

Breathing circuit - does it have carbon dioxide absorption or not? If so, is it fresh? Is the circuit intact, and does it work?

Airway equipment (Figure 1, Table 2) - Are there airways of various types and sizes - oropharyngeal, nasopharyngeal, endotracheal tubes, laryngeal masks? Are an endo-tracheal tube introducer and a bougie immediately available? Are there a syringe, clamp, tape, Magill's forceps, catheter mount available? Is there a way to insufflate the trachea with oxygen if the patient cannot be ventilated or intubated?

Can an emergency cricothyroidotomy be performed? Is there effective suction with handpieces and catheters?

Table 2 Airway Equipment

<table>
<thead>
<tr>
<th>Suction Device</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oral/Nasal Airways</td>
</tr>
<tr>
<td>Laryngoscopes</td>
</tr>
<tr>
<td>Endotracheal Tubes</td>
</tr>
<tr>
<td>Syringe/clamp/tape</td>
</tr>
<tr>
<td>Introducer/Stylet</td>
</tr>
<tr>
<td>Bougie</td>
</tr>
<tr>
<td>Magill's Forceps</td>
</tr>
<tr>
<td>Laryngeal Mask</td>
</tr>
</tbody>
</table>

Cricothyroid Insufflation Equipment

Cricothyroid Equipment

- Breathing equipment - Are there face masks of various types and sizes? What is the main ventilating system? Is there a self-inflating bag in reserve? Is the equipment for emergency decompression of a tension pneumothorax available? Is there a ventilator for long cases?

- Circulatory equipment - What intravenous equipment is there? - syringes, needles, catheters, fluids, ability to infuse under pressure, ability to warm intravenous fluids.

- Other equipment - What equipment is available to warm or cool the patient? What monitoring equipment is there that works and has been checked. Complete monitoring can be listed as follows, bearing in mind that some hospitals will have all of it, and some will have very little.

- Clinical monitoring by the anaesthetist, of the patient, the surgery and the equipment.

  - Pulse, colour, blood pressure, perfusion, skin feel.
  - Chest movement, breath sounds.
  - Pupil size, lacrimation
  - Temperature, urine output.
  - Pulse oximetry (the most useful electrical monitor)
  - Capnography (the second most useful electrical monitor)
  - ECG (the third most useful electrical monitor)
  - Airway pressure, tidal and minute volumes
  - Blood sugar, haemoglobin level, blood gases
  - CVP monitoring equipment
For children, is all the equipment of the appropriate type and size?

Drugs - There are so many, and the choice between them is often based on arguments which may be relevant in some environments and not in others.

Intravenous induction agents (Table 3) - thiopentone is still the commonest agent world-wide, challenged by ketamine in some places, propofol in others.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Typical Initial Dose</th>
<th>Clinical Onset</th>
<th>Clinical Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thiopentone</td>
<td>4-5 mg/kg</td>
<td>20-30 sec</td>
<td>5-10 min</td>
</tr>
<tr>
<td>Propofol</td>
<td>1.5-2.5 mg/kg</td>
<td>1-2 min</td>
<td>5-10 min</td>
</tr>
<tr>
<td>Midazolam</td>
<td>0.01-0.1 mg/kg</td>
<td>2-4 min</td>
<td>1-2 hrs</td>
</tr>
<tr>
<td>Diazepam</td>
<td>0.02-0.2 mg/kg</td>
<td>3-6 min</td>
<td>4-8 hrs</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>1.1-1.5 mcg/kg</td>
<td>1-4 min</td>
<td>2-3 hrs</td>
</tr>
<tr>
<td>Morphine</td>
<td>0.05-0.15 mg/kg</td>
<td>3-10 min</td>
<td>2-3 hrs</td>
</tr>
<tr>
<td>Pethidine</td>
<td>0.5-1.5 mg/kg</td>
<td>2-5 min</td>
<td>2-3 hrs</td>
</tr>
<tr>
<td>Ketamine</td>
<td>1-2 mg/kg</td>
<td>20-30 sec</td>
<td>5-10 min</td>
</tr>
</tbody>
</table>

Inhalational agents (Table 4) - ether and halothane are common in many parts of the world, enflurane, isoflurane, sevoflurane in others.

<table>
<thead>
<tr>
<th>Agent</th>
<th>MAC</th>
<th>Concentration used</th>
<th>Blood/Gas partition Coefficient</th>
<th>Oil/water solubility</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ether</td>
<td>1.92</td>
<td>2-15%</td>
<td>12</td>
<td>3</td>
</tr>
<tr>
<td>Halothane</td>
<td>0.76</td>
<td>0.5-3%</td>
<td>2.3</td>
<td>220</td>
</tr>
<tr>
<td>Enflurane</td>
<td>1.68</td>
<td>1-6%</td>
<td>1.9</td>
<td>120</td>
</tr>
<tr>
<td>Isoflurane</td>
<td>1.15</td>
<td>1-4%</td>
<td>1.4</td>
<td>120</td>
</tr>
<tr>
<td>Sevoflurane</td>
<td>2</td>
<td>1-6%</td>
<td>0.69</td>
<td>53</td>
</tr>
<tr>
<td>Nitrous Oxide</td>
<td>104</td>
<td>70%</td>
<td>0.47</td>
<td>2.2</td>
</tr>
</tbody>
</table>

* with 60% nitrous oxide. MAC is higher if no nitrous oxide is used

Hypnotics - diazepam remains common, but midazolam is more useful in anaesthesia because of its more rapid onset and shorter duration of action.

Opioids - morphine is still widely used, and pethidine less frequently. Fentanyl is increasingly used in anaesthesia because of its short duration of action.

Other analgesics such as paracetamol or indomethacin suppositories.

Muscle relaxants (Table 5) - Suxamethonium is still the choice for emergencies. Non-depolarising relaxants are now many, and may be short, medium or long acting, with specific advantages and disadvantages. Vecuronium, atracurium and rocuronium are rapidly overtaking pancuronium in many places. d-tubocurarine, alcuronium and gallamine are still used in some countries.
### Table 5 - Muscle Relaxants

<table>
<thead>
<tr>
<th>Drug</th>
<th>Initial Dose mg/kg</th>
<th>Approximate duration (min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>d-Tubocurarine</td>
<td>0.5</td>
<td>25-30</td>
</tr>
<tr>
<td>Alcuronium</td>
<td>0.3</td>
<td>20-25</td>
</tr>
<tr>
<td>Gallamine</td>
<td>1-2</td>
<td>20-30</td>
</tr>
<tr>
<td>Pancuronium</td>
<td>0.1</td>
<td>30-45</td>
</tr>
<tr>
<td>Vecuronium</td>
<td>0.1</td>
<td>15-20</td>
</tr>
<tr>
<td>Atracurium</td>
<td>0.5</td>
<td>20-25</td>
</tr>
<tr>
<td>Cisatracurium</td>
<td>0.15</td>
<td>20-25</td>
</tr>
<tr>
<td>Mivacurium</td>
<td>0.2</td>
<td>10-20</td>
</tr>
<tr>
<td>Rocuronium</td>
<td>0.6</td>
<td>20-30</td>
</tr>
<tr>
<td>Suxamethonium</td>
<td>1-1.5</td>
<td>3-5</td>
</tr>
</tbody>
</table>

Other essential drugs include atropine, neostigmine, adrenaline, ephedrine, an anti-hypertensive, a bronchodilator, a diuretic, an anti-emetic, and emergency resuscitation drugs (atropine, calcium, adrenaline, lignocaine) (Table 6).

### Table 6 - Resuscitation Drugs

<table>
<thead>
<tr>
<th>Drug</th>
<th>Recommended Dose</th>
<th>Average Adult Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adrenaline</td>
<td>0.01-0.05 mg/kg</td>
<td>0.5-1 mg</td>
</tr>
<tr>
<td>Atropine</td>
<td>0.02 mg/kg</td>
<td>0.6-1.2 mg</td>
</tr>
<tr>
<td>Calcium Chloride</td>
<td>0.2 ml/kg (10%)</td>
<td>5-10 mL</td>
</tr>
<tr>
<td>Lignocaine</td>
<td>1 mg/kg</td>
<td>10 mL 1%</td>
</tr>
</tbody>
</table>

Local anaesthetics - lignocaine, bupivacaine, ropivacaine, cinchocaine.

### Pre-Operative Assessment and Resuscitation

A systematic approach is best - it avoids overlooking important matters. For the patient requiring emergency abdominal surgery, with few exceptions, there is time to assess properly, and to resuscitate, before induction of anaesthesia. Most sensible surgeons understand this. Even in the few surgical emergencies where time to surgery is critical, the anaesthetist must still have essential information before proceeding.

Bear in mind that patients (and surgeons) do not tolerate unnecessary delays. If the patient needs investigations and/or resuscitation, organise it yourself, then you know it has been done properly. Don’t “leave it to someone else”. If surgery has to be delayed for resuscitation, agree on a time with the surgeon (see case insert).

There are some situations where the patient must go to theatre immediately - they include severe foetal distress, uncontrollable internal haemorrhage, rapidly expanding intracranial lesion (e.g. extradural haematoma). In these situations, history, examination, resuscitation have to be done "on the run" and with no delay. In most other situations, a short delay for resuscitation is best for the patient.

A good approach is to divide pre-operative assessment and resuscitation into two phases - initial (rapid), and definitive (when there is more time). In the history, essential questions are:

- When did you last eat or drink? (But regard these patients as having a full stomach anyway.)
- Have you any allergies?
- Are you taking any medications, smoking, drinking, using drugs or remedies?
- Have you had any problems with previous anaesthetics?
- Heart problems, chest problems, kidney or liver problems?
- Diabetes?
- Heartburn or reflux?
- Fits, fainting, or funny turns?
- Bleeding tendency?
- Pregnancy?
- Infectious disease? - especially HIV/AIDS, Hepatitis, Malaria, TB

In the physical examination, look particularly for evidence of
- Difficult airway
- Respiratory abnormalities
- Cardiovascular abnormalities

Investigations may not be available, or not available in the time frame. Haemoglobin, urea, creatinine, electrolytes, Chest X-Ray and ECG are still the most useful.

Investigations may be clinical, or laboratory. Clinical investigations are part of physical examination, and include the "bedside forced expiratory volume", measured with a spirometer, or by listening to rapid exhalation. Laboratory investigations should always be requested if they will help to identify a problem which can be corrected. Once ordered, they must be checked and acted upon. Once again, they may or may not influence a clinical decision to delay the operation, or to proceed.

Of the more commonly available investigations, Haemoglobin value must be interpreted in the context of the usual Hb of the population (which may be 8-9gm/dl in some areas, 12-13gm/dl in others) as well as in the context of bleeding or dehydration. A Hb of 8gm/dl in a bleeding or dehydrated patient may really be 5gm/dl when resuscitation is complete, and vascular volume is expanded, so blood transfusion may be indicated early.

Blood sugar (or urinalysis for glucose) should always be measured to allow correction in the diabetic, and to detect diabetic ketoacidosis masquerading as an abdominal emergency.

Urea and Creatinine and Electrolytes may be helpful, but should be interpreted in the context of the clinical picture, and information about whether the patient has pre-existing renal failure.

Elevation of urea and creatinine may simply indicate dehydration and poor renal blood flow, or it may indicate acute or chronic renal failure. Fluid resuscitation should proceed whatever the cause, to ensure renal blood flow is improved.

Serum sodium, potassium, chloride and bicarbonate may be "normal" or "abnormal". The first step in the acute abdominal emergency is again expansion of intravascular volume and fluid resuscitation. If renal function can be restored, the kidneys will correct the electrolyte disturbance.

Chloride and bicarbonate tend to balance each other - if one goes up the other goes down. Hypochloraemia (as in pyloric stenosis) will correct with normal saline infusion, but be made worse with Hartmann’s solution, because of the lactate, which is converted to bicarbonate. A low bicarbonate usually indicates metabolic acidosis due to poor perfusion, and corrects as the circulation improves.

Administration of bicarbonate is not often advisable, because it combines with hydrogen ions and results in formation of carbon dioxide which must be excreted by increased ventilation. Its acidosis-correcting effect is thus short-lived.

Arterial blood gases are the only accurate way of obtaining:
- PaO 2 (Oximetry is a substitute provided perfusion is good)
- PaCO 2 (End tidal CO 2 is a substitute but in the critically ill patient, there may be a wide gap between the ETCO 2 and the higher PaCO 2, not the normal 6mmHg)
- pH
- HCO 3 (which may differ from that measured with serum electrolytes)
- Identification of whether an acid-base disturbance is an acidosis or alkalosis, whether either is primarily metabolic or respiratory, and whether there is secondary compensation for the primary disturbance.

Chest X-Ray is often useful in patients with abdominal emergencies when history and examination are not clear cut, particularly in obese patients. Look carefully for pneumothorax, haemothorax, effusion, evidence of stomach or bowel in the chest, abnormalities in the lung fields (basal atelectasis is common), size and outline of the cardiac shadow.
ECG may indicate ischaemia, atrial or ventricular enlargement, abnormalities of electrolytes (as in the peaked T waves of hyperkalaemia), arrhythmias.

Assess the risk for this patient. Were they perfectly healthy before the emergency, or did they have mild systemic disease, significant systemic disease, or life-threatening systemic disease now complicated by an emergency?

Be aware of common conditions in the population which will influence resuscitation and anaesthesia, as well as postoperative care. These may include:

- Diabetes
- Ischaemic heart disease, cardiac failure, hypertension
- Valvular heart disease
- Asthma, chronic respiratory disease
- TB - especially of pleura and pericardium
- HIV/AIDS
- Malaria
- Anaemia
- Liver disease, renal disease

Identify, pre-operatively if possible, those patients who will benefit from close observation and care post-operatively in the High Dependency or Intensive Care Unit. You may be responsible for care of the patient there. If not, make sure the handover is good, and that you are available to help if there are problems.

**Resuscitation goes hand in hand with assessment**

- Airway problems such as in severe facial injury must be managed before induction of anaesthesia.
- Oxygen should always be given to the critically ill patient.
- Breathing problems such as asthma or pneumothorax must be treated before induction of anaesthesia.
- Circulation problems such as hypovolaemia, or cardiac tamponade must be treated before induction of anaesthesia.
- Other emergencies, such as hyperglycaemia and electrolyte or acid-base abnormalities must have treatment commenced before induction of anaesthesia.
- Consider the need for a nasogastric tube. Decide when to insert the urinary catheter.

Resuscitation must be aggressive before and during anaesthesia. The only excuse for induction prior to resuscitation is if the patient has a condition which cannot improve without surgery. This may include massive intra-abdominal haemorrhage. Even then, resuscitation must begin before anaesthesia is induced.

Which fluids should be used in resuscitation depends on the cause of the problem, and what is available. (Table 7).

In an adult with intra-abdominal bleeding, the choice is clearly blood and plasma expanders such as Haemaccel or Gelafundin or Gelafusin or Dextran, supported by crystalloids - normal saline or Ringer lactate (Hartmann’s) solution.

In a patient with intra-abdominal sepsis, the same approach may be needed, but blood transfusion will depend on the haemoglobin level once vascular volume has been restored. In an adult with bowel obstruction who is not shocked, saline or Hartmann’s solution may be adequate. In an infant with pyloric stenosis, saline is required initially, and Hartmann’s solution will make the hypochloraemic metabolic alkalosis worse.

What fluids to give, and how much, depends on the cause of the emergency. Every patient with shock is an opportunity to revise your cardiovascular pathophysiology.
Table 7 - Intravenous Fluids

<table>
<thead>
<tr>
<th>Fluid</th>
<th>Na+ mmol/L</th>
<th>K+ mmol/L</th>
<th>Cl- mmol/L</th>
<th>HCO3- mmol/L</th>
<th>Ca++ mmol/L</th>
<th>Mg++ mmol/L</th>
</tr>
</thead>
<tbody>
<tr>
<td>N/Saline</td>
<td>152</td>
<td>-</td>
<td>154</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Hartmann’s</td>
<td>131</td>
<td>5</td>
<td>111</td>
<td>29*</td>
<td>2</td>
<td>-</td>
</tr>
<tr>
<td>4% Dextrose in</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>N/5 Saline</td>
<td>31</td>
<td>-</td>
<td>31</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>5% Dextrose</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* as lactate

Tissue perfusion of the whole body depends on an adequate cardiac output. Cardiac output depends on:

- **Myocardial contractility**, which is influenced by
  - End diastolic ventricular volume
  - End systolic ventricular volume
  - Myocardial integrity

- **End diastolic volume**, or the volume of each ventricle before it contracts, is influenced by
  - End systolic volume
  - Preload

- **End systolic volume**, or the volume of each ventricle at the end of contraction, is influenced by
  - End diastolic volume
  - Afterload

- **Preload - venous return to the atria** depends on blood volume, vascular capacitance (matching of blood volume to vascular capacity), posture, venous valves, limb muscle activity, intrathoracic pressure changes, functioning cardiac valves, normal atrial contraction, and a reasonable heart rate to allow time for ventricular filling.

- **Afterload - ejection of the stroke volume into the aorta** is influenced by the ability of the arterial bed to receive the volume, so that vasoconstriction requires extra cardiac work to generate the pressure required to eject the blood.

- **Myocardial integrity** depends on the cardiac muscle having glucose and oxygen to allow it to function properly. It will be impaired if there is myocardial ischaemia, some electrolyte imbalances, or if there are toxins (from sepsis) affecting it, or if it is exposed to high concentrations of some anaesthetic agents (intravenous or inhalational).

In an abdominal emergency, the main problem resulting in poor tissue perfusion may be

- **Hypovolaemia** (as in haemorrhage)
- **Hypovolaemia plus vasodilatation** (as in sepsis)
- **Hypovolaemia plus vasodilatation plus myocardial depression** (as in sepsis).

In all cases, apart from giving oxygen, the most important thing to do is to correct the hypovolaemia, start antibiotics, then review and rethink. In sepsis, use of a “vasopressor” may be wise after correction of the volume deficit. Although there are several available, the cheapest and most useful is the catecholamine adrenaline. If the patient is moribund, intermittent doses of 0.1-0.5mL of 1:10,000 adrenaline may buy time until an infusion of 3-12mcg/minute can be set up (3mg adrenaline in 50mL normal saline run at 3-12mL/hour).

Note that in a resuscitated patient, it may take several hours for urine output to improve, even though the perfusion, blood pressure and pulse rate improve rapidly. In every patient, monitor the effects of the drug used to check that the desired effects are being achieved.
How fast fluids should be administered depends on the estimated deficit and the time available to ensure the circulatory volume is adequate before induction of anaesthesia. Always use large bore IV cannulae - more than one if necessary.

The aim is to have a patient who is conscious, pink, well perfused, with a reasonable pulse and blood pressure prior to induction. Particular care is required in the very young and the very old.

Case Insert
A 30 year old male has been admitted with peritonitis, thought to be due to bowel perforation from typhoid, present for 3 days. He is shocked, with a temperature of 38°C, a pulse of 120/minute, BP 70 mmHg systolic, poor nail bed capillary return, respiratory rate 30/minute, confused. There are no facilities for immediate investigations of any sort. Urinary catheterisation results in 20mL of concentrated urine. The surgeon wants to operate immediately. The anaesthetist does not say "Yes", or "call me when the patient is resuscitated". The anaesthetist does ask the surgeon to assist in resuscitation following the ABC sequence, planning to resuscitate with oxygen, IV fluids, and administer antibiotics.

This patient could be deficient in fluids to the extent of at least 8-10 litres or more, (2 litres per day x 3 days of maintenance fluids plus fluid lost by vomiting/diarhoea, plus fluid pooled in the bowel and peritoneal cavity). Induction of general anaesthesia in this state will probably cause death. The first priority is restoration of intravascular volume with a colloid such as Haemaccel/Gelofundin/Gelofusin or Dextran given rapidly, until the pulse rate is down, the blood pressure is up, nail bed perfusion has improved, and the patient’s mental state has improved. If colloids are not available, use a crystalloid such as normal saline or Hartmann’s solution. Higher volumes of crystalloids will be required because of their rapid distribution throughout the extracellular fluid space. Once the patient has acceptable vital signs and looks better, run saline or Hartmann’s solution rapidly while getting ready for theatre. If you do the resuscitation, the patient may be ready for induction of anaesthesia in 1-2 hours. If you delegate the resuscitation and wait for a phone call, the patient may never survive to get to theatre.

Preparation of the Patient for Theatre
Two questions which arise after assessment of the patient has been completed, and resuscitation is underway, are what about fasting and what about premedication?

In an abdominal emergency it is always assumed that the stomach is full, and that an emergency rapid sequence ("crash") induction and intubation of the trachea will be carried out. There is no need to fast, but there is a need to decide whether emptying the stomach by nasogastic tube is advisable - as in bowel obstruction, when vomiting or regurgitation of large amounts of fluid may result in aspiration or hypoxia. Pre-medication should be restricted to use of opioids intravenously for analgesia, and atropine if ether or ketamine are to be used. Hypnotics should not be given, because they increase the risk of regurgitation and aspiration in these patients. Antiemetics will not be effective. Antacids and H2 antagonists are most effective for the emergency patient with an empty stomach, which is rare. Make sure that any resuscitation measures commenced are continued up to the time of induction of anaesthesia.

Induction of Anaesthesia
There are two phases, the ‘countdown’ to induction, and induction itself. The ‘countdown’ is the short period of checking that everything is ready, and nothing has been missed. (Table 8). This is when the patient is on the operating table, the assistant is ready to do anything required, including hand you the sucker, apply cricoid pressure reliably and effectively, and tilt the table head down on request. The anaesthetic machine, equipment and drugs have been prepared and checked. The intravenous line(s) is running well. The surgeon is scrubbed and the nurses waiting. The monitors are checked, and readings noted. 100% oxygen has been administered for 5 minutes. Now it is time to start the induction sequence, informing the patient that they will feel sleepy shortly, and that pressure will be applied to their throat (Table 9).

The intravenous induction agent is given slowly until the patient does not respond, bearing in mind that the circulation time may be slow in these patients. Cricoid pressure is applied suxamethonium is given, and tracheal intubation performed as soon as the fasciculations start to fade. The cuff is inflated, and the patient ventilated with a few breaths of 100% oxygen while checking the position of the tube. The tube is then secured.

How do you know the tube is in the trachea? (Table 10). Because you saw it pass through the vocal cords, heard bilateral breath sounds, with no noise over the epigastrium, and the chest moved uniformly up and down. What else
is useful? Capnography is the gold standard, disposable colour-change discs are the next best. Without either of these, aspiration of the endo-tracheal tube with a large syringe will reveal easy aspiration of air if the tube is in the trachea, with a vacuum if it is in the oesophagus.

Table 8 Pre-Induction ‘Countdown’

Patient
Surgeon
Assistant to Anaesthetist
Machine Check
Airway Management
Breathing Equipment
Circulation Equipment
Anaesthetic drugs are drawn up
Resuscitation drugs are available
Intravenous
Pre-Oxygenation
Vital Signs
Monitors

Table 9 Induction Sequence

Give 100% oxygen
Complete pre-induction ‘Countdown’
Assistant ready
Thiopentone +/- fentanyl
Suxamethonium
Cricoid pressure
Endotracheal tube insertion
- Cuff up
- Check position
- secure tube
Non-depolarising relaxant
O 2 /gas/vapour
Check vital signs/monitors
Check patient safety

If at this stage you are unable to intubate or ventilate the patient, tell the surgeon, and start the protocol you worked out before you started. Maintain oxygenation, maintain cricoid pressure and follow the sequence shown in Table 11.

Table 10 Is the Tube in the Trachea

See it pass through the cords
Chest moves uniformly
Hear bilateral breath sounds
No noise over epigastrium
Capnography trace
Table 11 Failed Initial Intubation

Call for help
Maintain cricoid pressure
Ventilate with 100% O2

If you can ventilate
- Reposition head
- Manipulate larynx
- Suction larynx
- Use introducer or bougie
- Reintubate with smaller

If you can’t ventilate
- Consider LMA
- Consider cricoid insufflation
- Consider cricothyroidotomy
- Consider waking patient up

Maintenance of Anaesthesia

Maintenance of anaesthesia (Table 12) may be achieved with nitrous oxide, oxygen and a volatile agent. If there is no nitrous oxide or it is contra-indicated, an air/oxygen mixture and volatile agent can be used. If there is no oxygen, just air and volatile agent, bearing in mind that the amount of the anaesthetic agent required will be higher than if it is used with nitrous oxide. If there is no air, oxygen and volatile agent can be used. A nondepolarising muscle relaxant and intermittent positive pressure ventilation allows the best conditions for the surgeon. If there are no relaxants, controlled or assisted ventilation will still assist the surgeon.

Table 12 Maintenance of Anaesthesia

Maintain Anaesthesia
- Agents/gas mixture
- Opioids
- Relaxants
- Monitor
- Vital Signs

Monitor
- Blood loss
- Fluid/blood replacement
- Urine output

The maintenance phase requires observation and monitoring of the patient, and of the surgery, with particular attention to fluid and blood loss. If major surgery is proposed, or if the patient was dehydrated or hypovolaemic, measurement of urine output is a good guide to renal perfusion. Keep a careful record of anaesthetic agents, monitored variables, fluid and electrolyte balance.
Potential anaesthetic problems that may occur are the development of high or low airway pressure, desaturation of haemoglobin, abnormalities in the capnometry trace, hypotension, hypertension or arrhythmias. For each scenario, have a plan of how to find the cause of the problem in a logical way. (Table 13).

### Table 13 Checking Problems

<table>
<thead>
<tr>
<th>High Airway Pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Misplaced airway/ETT</td>
</tr>
<tr>
<td>- Blocked airway/ETT</td>
</tr>
<tr>
<td>- Kinked airway/ETT</td>
</tr>
<tr>
<td>- Bronchospasm</td>
</tr>
<tr>
<td>- Tension pneumothorax</td>
</tr>
<tr>
<td>- Sticking valve</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Low airway pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Where is the leak?</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Desaturation of Haemoglobin</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Oxygen supply failure</td>
</tr>
<tr>
<td>- Oxygen delivery failure</td>
</tr>
<tr>
<td>- Poor ventilation</td>
</tr>
<tr>
<td>- Poor perfusion</td>
</tr>
<tr>
<td>- Artefact</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Abnormal CO2 trace</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ventilator problem</td>
</tr>
<tr>
<td>- Circuit problem</td>
</tr>
<tr>
<td>- Circulatory problem</td>
</tr>
<tr>
<td>- Air embolism</td>
</tr>
<tr>
<td>- Artefact</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Hypotension - identify cause and treat</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension - identify cause and treat</td>
</tr>
<tr>
<td>Arrhythmias - identify cause and treat</td>
</tr>
</tbody>
</table>

**Monitoring**

The most important monitoring of the patient is clinical, including pulse, blood pressure, colour, respiration, pupil size, lacrimation, in addition to monitoring the surgical field, blood loss, urine output, fluid input. Heart sounds are useful to monitor particularly in children.

The next important set of instrument monitors are pulse oximetry, end tidal CO2 monitoring, ECG and temperature. If available, CVP monitoring may be a useful guide, particularly in the patient who you think has had adequate fluid/blood replacement, but who remains hypotensive. Supported by a high CVP reading, this may be an indication for adrenaline infusion rather than more fluid, provided all other causes of hypotension have been looked for (e.g. pneumothorax, excess anaesthetic agent).

Other forms of monitoring in the critically ill patient might include an arterial line for BP and blood gas sampling, and occasionally a pulmonary artery catheter, which may show that despite a high CVP, the left atrial pressure, as reflected by the pulmonary capillary wedge pressure, is low.

Neuromuscular function monitoring is helpful in those patients who do not breathe well after reversal of muscle relaxants.
In situations where they are available, monitoring of inspired and expired oxygen, nitrous oxide and volatile agent should be used. Airway pressure, tidal and minute volume measurements likewise should be used if available.

Reversal of Anaesthesia

The end of surgery is the beginning of the next challenging period for the anaesthetist. It requires planning, like it did before induction. A “countdown” (Table 14) ensures that the sequence of timing of cessation of the volatile agent, reversal of the muscle relaxant with atropine and neostigmine, return of spontaneous ventilation, suction of the mouth and pharynx, and extubation of the patient occur smoothly (Table 15). Again, the assistant must be ready to start suction, and tilt the table if required.

Table 14 Reversal ‘Countdown’

Check Equipment
Check drugs
Assistant ready
Turn off agents
Give 100% oxygen
   Suction
   Reverse relaxant
Check Observations
Wait for adequate breathing
Wait until patient wakes up
   Extubate
   Give 100% O2 by mask
DO NOT LET THE PATIENT MISS A BREATH

Table 15 Reversal Sequence

Check
- Vital signs/monitor
- Surgeon is finishing
   - Assistant ready
- Time of last dose of relaxant
   - Signs of reversal
Check “Countdown” complete
   Extubate
   Turn patient on side
Check airway is clear
   100% O2
DO NOT LET THE PATIENT MISS A BREATH
Check vital signs/monitors
ALL HANDS to move patient
Transfer to recovery
A final check of observations, and the patient’s ability to maintain their airway, ventilation and oxygenation, and movement to the bed or trolley and transfer to Recovery can proceed. But a number of things can go wrong at this stage. There may be inadequate muscle relaxant reversal, and more reversal agent may be required, or extubation may have to be delayed; extubation may be followed by regurgitation or vomiting and aspiration; there may be laryngeal spasm. On the circulatory side, hypotension may occur while attention is concentrated on airway and breathing. A plan for each of these events must have been made, so that no time is lost in detecting and correcting the problem.

**Recovery Room Care**

Care in the Recovery Room must equal that during anaesthesia until the patient is capable of looking after their own airway and breathing, and is fully conscious. Again, use a systematic approach (Table 16). Any problems must be identified and treated rapidly (Table 17).

### Table 16 Recovery Care

- Check vital signs/monitors
- Check level of consciousness
  - Continue oxygen
  - Check wound
  - Check urine output
- Check respiratory rate, sedation, pain score
- Check temperature
- Give analgesics as required IV
- Check fluids and IV sites

### Table 17 Some recovery Problems

- Inadequate breathing
- Regurgitation/vomiting/aspiration
- Laryngeal spasm
- Hypotension
- Not waking up

The patient in Recovery should continue to receive oxygen, have continuous monitoring of airway, breathing and circulation, and be given analgesia as required. Specific problems require a plan. If the patient fails to breathe adequately, is it due to inadequate reversal of relaxants, to the persistence of anaesthetic agents and opioids? Have they continued to bleed or lose fluid since the anaesthetic finished, and become hypovolaemic? If the patient fails to wake up, is it because of the drugs given, hypoxia, carbon dioxide retention, hypoglycaemia, hypothermia, or a medical complication?

**Postoperative Care**

The anaesthetist is often the best resource a surgeon has to advise on post-operative problems such as pain relief (Table 18), management of nausea and vomiting, fluid and electrolyte replacement. Get in the habit of visiting all emergency patients in the ward. You may be able to help, and you can make a note of any problems recorded on your anaesthetic record or the recovery record, as well as picking up anything that developed later which the surgeon believes may be due to the anaesthetic. You can also encourage early mobilisation and chest physiotherapy to minimize postoperative complications such as atelectasis, pneumonia, and deep venous thrombosis.
Table 18 Post Operative Pain relief

- Opioids
  - Titrate intravenously to start
  - Continue SCI or IMI regularly or IV infusion
  - Wean to simple analgesics
- Regional - epidural
  - Monitor pain on a 0-10 scale
  - Top-up before mobilisation
- Check for side effects
  - Respiratory depression
  - Sedation
  - Nausea/vomiting/itching
  - Confusion/hypotension
  - Urinary retention

Regional Anaesthesia
Occasionally, there may be a surgeon, an anaesthetist with only facilities for regional anaesthesia, and a patient requiring emergency abdominal surgery who cannot be moved to another hospital. Can anything be done with regional anaesthesia?

The options available are not ideal forms of anaesthesia for emergency abdominal surgery, but if resuscitation is carried out and the same principles followed as have been described above, possibilities include:

- Spinal anaesthesia
- Epidural anaesthesia
- Abdominal field block
- Para-vertebral block
- Splanchnic block

Spinal and epidural blocks have been described superbly in previous issues of Update (see Further Reading). They must not be used in patients who have not been fully resuscitated. Abdominal field block is best carried out by paravertebral intercostal block, first described by Sellheim in 1906, or paravertebral block, described by Kappis in 1912. Abdominal field block was first carried out by Schleich in 1899. Posterior splanchnic block was described by Kappis in 1919. These blocks have significant complications, and should only be attempted by those with excellent anatomical knowledge and technical skills.
INDUCTION OF ANAESTHESIA IN PAEDIATRIC PATIENTS

Introduction
Induction of anaesthesia in children is achieved with broadly the same anaesthetic agents and techniques as are used in adults. However, there are some important differences in the pharmacology of the agents available when comparing adults and children. Also, the technical difficulties that are associated with small size and the psychological and behavioural issues due to immaturity may make induction of anaesthesia more challenging in the child compared to the adult.

Paediatric anaesthesia has been the subject of this journal before.1 Analgesic methods in children have also been discussed.2 In this short paper, the pharmacology of the induction agents will be covered. The different methods available for inducing anaesthesia will be discussed and the merits of each method compared. Techniques that are specifically designed to overcome difficult situations relevant to paediatric anaesthesia will be discussed.

There are several methods of anaesthetic induction: Gaseous induction, breathing a mixture of volatile anaesthetic agents until loss of consciousness is achieved; Intravenous induction, where an anaesthetic drug is injected intravenously in a dose sufficient to produce unconsciousness; Other, where an induction agent is given by a non-intravenous route, generally orally, rectally or intramuscularly, to produce loss of consciousness.

Pharmacology - the intravenous agents
Sodium Thiopentone. First introduced in the 1930's, this barbiturate has been the mainstay of intravenous induction ever since. It is also known as pentothal or Nembutal. It is supplied as a yellow powder mixed with sodium carbonate and is dissolved in water before use. Solution in water results in an alkaline pH. The concentration in water is important as pain free injection is only reliable with a dilute solution (2.5% or 25mg/ml or less). The solution may result in severe tissue necrosis if injected extra-vascularly and this is worse with the more concentrated solutions. The induction dose of thiopentone is between 4-6 mg/kg in adults and 5-7 mg/kg in infants and children.

Induction of anaesthesia is rapid and generally accompanied by minimal excitatory effects such as involuntary movement or hiccupping. Thiopentone is protein bound and highly lipid soluble. It's effect on the central nervous system, unconsciousness, is terminated by redistribution of the drug which results in recovery of consciousness about 5 minutes after induction by a single dose. If repeated doses are administered, recovery may be significantly prolonged. The drug is metabolized by the liver but the efficiency of this process has little bearing on the anaesthetic duration of action. Children handle thiopentone slightly differently from adults. The induction dose is higher. The elimination half time is reduced and this means that "hang-over" after thiopentone induction, is much less of a problem in children than in adults. When used clinically, anaesthesia is induced rapidly after injection. There is generally a short pause in respiration, but this rarely lasts more than a few seconds. Respiration then resumes and a volatile agent may be introduced whilst the patient is spontaneously breathing. Heart rate generally rises slightly on injection but there is vasodilation and a drop in cardiac output. This is clinically significant in hypovolaemic patients and those with intercurrent medical conditions but in otherwise healthy patients, is well tolerated. Cardiovascular compromise is less marked than with propofol. Hypersensitivity (allergy) is rare but generally very serious. The risk of anaphylaxis is quoted at 1:50,000 administrations but may carry a 50% mortality. The major specific contraindication is porphyria.

Propofol is a non-barbiturate intravenous anaesthetic agent introduced in the 1980s. It is presented as an aqueous solution in soya oil and egg phosphatide. In a dose of 2.5-4mg/kg, it rapidly induces anaesthesia. In this dose, excitatory movement is common and it is now common practice to use a higher dose in unpremedicated children. Typically, 4mg/kg is administered as a bolus, followed by aliquots of 0.5-1mg/kg to allow a smooth transition from propofol anaesthesia to a vapour based anaesthetic. Even in higher doses, there are more excitatory and involuntary movement than with thiopentone. Unlike thiopentone, repeat doses may be given without unduly affecting the quality of postoperative recovery. Indeed, it is used as a sedative for medium to long term use in the intensive care unit (not in children). An induction dose causes a more prolonged pause in respiration than thiopentone. With higher induction doses, this pause becomes longer and in clinical use, apnoea is frequently produced with this agent. Airway reflexes are depressed after an induction dose and it is said that airway instrumentation is facilitated more using this drug than with alternatives. The cardiovascular effects are dose dependent but a reduction in blood pressure is seen. In the hypovolaemic, this may be profound and dangerous. This effect is greater than with equivalent doses of thiopentone.
Although less irritant than thiopentone in the event of extra-vascular injection, the chief disadvantage to the use of this agent in children is pain upon injection. This pain is quite severe and detracts from a smooth induction. It may be alleviated, but not prevented, by the co-administration of lignocaine in a dose of 1mg lignocaine/ml of propofol 1% solution. Propofol infusion syndrome may result if propofol, in high doses, is infused over long periods of time in children who are critically ill. Whilst the mechanism of this syndrome is not completely clear, it appears that this is a safe drug for use as an induction agent in children but it is not licensed as a intravenous sedative in children. In the UK, propofol costs three times more than equivalent dose of thiopentone.

Etomidate is a non-barbiturate induction agent that is used in doses of 0.3-0.4 mg/kg. It's use results in less cardiovascular depression than thiopentone and there is little or no depression in the respiratory rate or depth. It is associated with considerable involuntary movement after injection and this makes induction much less "smooth" than with other agents. Etomidate is associated with pain on injection. Etomidate inhibits the synthesis of steroids by the adrenal gland and this finding has been used to explain a high mortality noted when this agent was used for sedation of young children on intensive care. Concern over it's inhibition of steroid synthesis, pain on injection and practical considerations relating to movement on induction have resulted in unpopularity of this drug for paediatric induction.

Ketamine is a non-barbiturate intravenous anaesthetic with many unusual and useful properties. Although anaesthesia is induced after an 2mg/kg intravenous injection, the presence of movement, opening of eyes and maintained respiration mean there is no clear "end-point" and it appears that induction is more prolonged than with thiopentone. However, there is preservation of heart rate and blood pressure at normal or supra-normal levels. Respiration is maintained at a higher rate and tidal volume. There is some preservation of airway reflexes during anaesthesia with this agent. The protective airway reflexes and increased respiration has lead to the popularity of this drug in circumstances where it may be used as a single agent, perhaps with limited access to anaesthetic equipment.

However, it must be acknowledged that with overdosage, airway reflexes may be lost and respiration depressed and oxygen must always be available for it's safe use. An advantage to this drug is the versatility in the manner in which it may be given. Ketamine may be administered via the intravenous, intramuscular, rectal and oral routes. Drawbacks to it's use are excessive salivation and unpleasant dreaming. Excessive salivation may be improved with the use of an antisialogogue such as atropine. The dreaming may be reduced by co-administration of a benzodiazepine. Ketamine is relatively inexpensive.

Benzodiazepines. Midazolam and diazepam injection have been used as induction agents. The dose of each member of this class of drugs is very variable. Thus a dose of 0.05 to 0.5mg/kg of midazolam may be required to induce sleep. The time to peak effect is much longer than other induction agents and most anaesthetists find the best use for this class of drugs is as a premedicant. As a pre-medicant, midazolam is widely used. It is given orally at a dose of 0.5-0.75mg/kg. It rarely produces deep sleep but renders a child placid and co-operative. Further it provides useful amnesia. Studies have demonstrated that midazolam may be used as premedication before day case surgery without delaying discharge.

Pharmacology - volatile agents used for gaseous induction.

Ether. The original volatile anaesthetic agent and still much in use world-wide, the high solubility and irritant nature of this agent means it is not an easy induction method in children. Due to the difficulties encountered in obtaining other drugs and equipment, it is still used as a sole anaesthetic in many places but will not be discussed further here.

Halothane was introduced in the 1950s and rapidly became popular for maintenance and volatile induction of anaesthesia in both adults and children. It is administered through a dedicated vaporizer into a carrier gas. It has a MAC of 1.1% in infants and 0.6 in the elderly. The smell is non-irritant and not unpleasant.

The most common method of inducing anaesthesia with halothane is to start with the patient breathing the carrier gas which might be a mixture of oxygen and air or nitrous oxide. Halothane is introduced at 0.5% and then the patient breathes 5 normal breaths, the concentration is increased by 0.5% for another 5 breaths until 5% halothane is reached. Once unconsciousness is produced, the concentration may be reduced to an appropriate level. Halothane is a respiratory depressant and tidal volume is reduced. Respiratory rate may actually be increased a little during halothane anaesthesia but the response to hypoxia or hypercarbia are attenuated. However, these effects are to a lesser extent than with other volatile agents with the exception of ether. The principal disadvantage of halothane is its potentiation of the arrhythmic effects of catecholamines on the myocardium. Arrhythmias, particularly ventricular arrhythmias, are more common with this agent than with other volatile agents. Hypercarbia
causes release of catecholamines from the adrenal gland and the combination of mild airway obstruction, hypercarbia and halothane are a frequent cause of arrhythmia under anaesthesia. Usually, these are benign and respond to correction of the airway obstruction but in the presence of administered adrenaline (by sub-cut injection) these arrythmias may be dangerous. Post exposure hepatitis has been reported with halothane and extensively investigated. Its occurrence in children is not clear but is certainly very rare indeed.

Enflurane and isoflurane are both more pungent than halothane and have no advantages for gaseous induction of anaesthesia

Sevoflurane has been used in Japan since the 1970s. It is a volatile agent with a MAC of 2.3 in infants and 1.8 in adults. Its major advantage is that it has a smell which is non-pungent and it is possible to induce anaesthesia with high concentrations from the outset. It has no arrythmogenic effect. When compared to halothane, higher concentrations may be used earlier in induction, without complaint. Therefore, it appears to cause a swifter onset of anaesthesia. A disadvantage is that it is a more potent respiratory depressant than halothane and therefore, breath holding may occur before a truly deep stage of anaesthesia is reached. For this reason, some anaesthetists prefer halothane to sevoflurane when performing gaseous inductions for airway obstruction. The other major disadvantage to this agent is its high cost.

Induction of anaesthesia

Gaseous induction of anaesthesia. Anaesthesia is commonly induced in infants and children by means of a "gas induction." This is less commonly the case with adults, leading some anaesthetists to be unfamiliar or lacking in confidence with this method. Children, understandably, are reluctant to have a "needle" to put them to sleep. Many are aware of an alternative and will prefer this method. Infants may be very hard to cannulate prior to an intravenous induction, meaning that a gas induction becomes preferable. In nearly all cases, anaesthesia will be induced without an intravenous cannula in place and intravenous drugs will be hard to administer, particularly if the anaesthetist is working single handed.

Neonates may lie on the operating table and breathe from an anaesthetic mask attached to a T piece, or similar, low resistance anaesthetic circuit. Older children will, if adequately informed, frequently behave very well and will lie on the operating table and accept a gas induction. Between these age groups, the skill of the anaesthetist must be fully employed to ensure a smooth induction. Infants and young children are often very reassured by the presence of a parent in the anaesthetic room. This may not be possible in some circumstances but if feasible, pays dividends. With the presence of a parent, the child may receive a cuddle whilst having a gas induction. An older child may be persuaded to co-operate by a parent. Various games may be employed to distract children enough for them to receive a gas induction. "Blowing up the balloon" will be familiar to most anaesthetists and is a very effective way of persuading children to "breathe the gas." Another useful technique is to use a strong smelling food substance and rub it in the face mask. Orange peel is a useful way of disguising anaesthetic gases. If rubbed on the mask, a "guess the fruit" game can be enjoyed whilst the child goes to sleep. Clear plastic face masks alleviate the claustrophobia associated with the black rubber face masks. If halothane is used, care should be taken to move up the concentrations incrementally, taking plenty of time to allow the child to breathe comfortably. Increasing the concentration too fast results in coughing. Many anaesthetists prefer to use 70% nitrous oxide before adding halothane as this means the patient is already partially anaesthetized before smelling the halothane. Sevoflurane is more forgiving in this respect. Once the patient is asleep, most anaesthetists switch to 100% oxygen as a carrier gas.

Once the child is asleep, any parents present should be invited to leave. A pulse oximeter may be applied if it has not already been positioned. The child should be disturbed as little as possible. Once asleep, the patient goes through an excitatory phase. If the child is moved about, for instance, to remove clothing, this is often the stimulus that provokes airway reflexes. The anaesthetist should continue holding the face-mask and child's airway, maintaining a clear airway and good ventilation using oxygen and a high concentration of volatile anaesthetic agent, until a deeply anaesthetised state is reached. At this point, the child may be moved to insert an iv cannula, to undress them, to apply other monitoring and to facilitate surgery. If an iv cannula is needed, this is the time to insert it. If a tourniquet is used, the insertion of an iv cannula may be achieved one handed whilst the anaesthetist holds the face mask. However, with harder subjects, another pair of hands, to hold the airway or perhaps, to cannulate the patient, will be invaluable.

The obvious question is what to do about any adverse events that occur before an iv cannula is inserted. The answer is that, with the exception of airway obstruction, all other problems are exceptionally rare. With the anaesthetist holding the patient's airway, he is ideally placed to diagnose and treat airway obstruction as it occurs.
Hypoventilation is noted by decreased excursion of the breathing reservoir bag. Airway obstruction may be revealed by noisy breathing or increased work of breathing (increased chest excursion with decreased bag excursion). Normally, correcting the position of the patient's head corrects hypoventilation. Noisy breathing is often due to upper airway collapse during expiration and a small amount of continuous positive airway pressure (CPAP) will resolve it. This is generally applied by keeping tension on the reservoir bag with one hand. If hypoxia occurs, as evidenced by the pulse oximeter trace or by cyanosis, check that a maximum concentration of oxygen is being given.

Increase the CPAP. Occasionally, an oro-pharyngeal airway is helpful but care should be taken that this is not inserted at too light a plane of anaesthesia. It is rare that serious laryngospasm can not be overcome with patience, CPAP, 100% oxygen and correct positioning. If the situation does not resolve consider other causes of airway obstruction and consider applying suction to the pharynx to clear any secretions which may be lying around the larynx. If it becomes necessary to paralyse the patient, after they are asleep, but before an intravenous cannula may be inserted, remember that suxamethonium may be given intramuscularly (5mg/kg) and will work within 2-3 minutes.

Many anaesthetists prefer to intubate patients once they are deeply anaesthetized with a volatile anaesthetic agent alone. It has been advocated that suxamethonium may be given intramuscularly into the tongue. I have no personal experience of this. My feeling is that it might render a difficult airway worse by adding intra-oral bleeding to the clinical picture. I have no evidence for or against this route being any faster, or slower, than into any other muscle.

There are some circumstances when inhalational anaesthetic induction is not the method of choice. If a child already has a cannula in situ, perhaps for maintenance fluid therapy, then it is more appropriate to use this cannula. Many children express a preference for intravenous anaesthetic induction. Also, there are numerous occasions where a rapid sequence induction is indicated and here, inhalational induction is completely inappropriate.

**Intravenous induction**

The main problems with intravenous induction are pain on insertion of the cannula, a natural aversion of children to "needles" and difficulty in insertion. These are all relevant to adults but here we may reason with our patients and explain why it is necessary and why the cannula may be hard to place.

Children as young as five may well understand the reasons behind needing a cannula and may even understand that sometimes it is not easy to insert them and a second go might be required. Whatever the reasoning powers of the child, the whole process may be made much pleasanter by the application of topical anaesthesia to prevent the child feeling the cannula needle. EMLA (eutectic mixture of local anaesthetics) takes about one hour to become effective.

If placed over a cannulation site for an adequate amount of time, it is very effective. Amethocaine gel works faster. Unfortunately, these drugs are not uniformly available and sometimes, the only sensible plan is to explain why a cannula is needed and to use the smallest gauge possible. If nitrous oxide is available, the child may breathe a mixture of nitrous oxide and oxygen whilst the cannula is inserted but this technique often seems to combine the worst aspects of both intravenous and inhalational techniques, the child getting a "nasty mask" and a "horrible needle!"

Insertion of a venous cannula may be easy if the veins are obvious. Sometimes, this is a difficult procedure. The task is harder when the child has a large amount of subcutaneous fat, a common situation in toddlers. Veins become smaller in cold, dehydrated and frightened children. A warm, well hydrated, comfortable child should be our aim and parental presence or pre-medication may well help.

After insertion of an intravenous cannula, suitable monitoring can be attached and an intravenous induction agent injected. The choice of agent is described above but in a healthy child, the normal choice is between sodium thiopentone and propofol. Propofol undeniably results in less "hang-over" in the postoperative period. However, after one hour, this difference between sodium thiopentone and propofol becomes very subtle in children. Pain on injection is a considerable problem, especially when we have gone to such lengths to secure painless venous access.

Therefore, unless immediate post-operative discharge is needed, my preference is still for sodium thiopentone. This drug is injected as a single bolus of 5-6 mg/kg, the child painlessly goes to sleep and after the briefest pauses, begins to breathe. Maintenance with a volatile agent may then be substituted without having to recourse to a period of positive pressure ventilation, with a bag and mask, as generally is the case when propofol is used.
Rapid sequence induction

The indication for a rapid sequence induction is the same in adults and children. If a risk of aspiration of gastric contents is foreseen, a rapid sequence induction should be performed.

The procedure for this is the same in children as in adults. A working intravenous cannula is mandatory. The patient should be monitored and positioned on a tilting trolley with suction readily available. Oxygen is administered via a close fitting mask for 3 minutes and anaesthesia induced. As the induction agent works, an assistant applies cricoid pressure to the cricoid ring, with one hand supporting behind the patient's neck. This manoeuvre seals the oesophagus and prevents material from the stomach and oesophagus reaching the pharynx. The traditional agents are sodium thiopentone 5mg/kg and suxamethonium 2-3 mg/kg.

In practice, this procedure presents several problems and it is rare to achieve such good pre-oxygenation in a child as with an adult. Good rapport and explanation works for middle sized children but in the younger or less co-operative, three or four good screams into the oxygen mask is often all that can be managed. In this technique, an attempt is made to pre-oxygenate the child pre-induction. The thiopentone is administered and sleep induced. Cricoid pressure is applied and the mask closely applied. The child should take a breath quite soon after the thiopentone and once this is seen, suxamethonium administered. The suxamethonium is effective more quickly than in adults at this dose.

A difficult situation is the child with a full stomach, needing emergency surgery, who can’t be cannulated. Although not ideal, I think the most practical way forwards here is to induce anaesthesia by volatile induction with the patient in the lateral position. Once anaesthesia is induced, it should be easier to secure intravenous access, apply cricoid pressure, turn the patient supine and perform intubation.

Other means of induction of anaesthesia

In rare instances, induction of anaesthesia is most appropriately conducted using ketamine. Ketamine may be given intramuscularly using a tiny needle and then reliably induces anaesthesia if used in a dose of 7.5mg/kg. Anaesthesia onset takes about 5 minutes. The intramuscular route seems more reliable than the oral route, for which, the dose range quoted in the literature is very wide.

Conclusion

The debates over which drugs and which methods are "best" to use to anaesthetize infants and children are popular at meetings of paediatric anaesthetists. The fact that these debates occur indicates that there are no answers. The skilled, confident anaesthetist, who is prepared to react flexibly and to adopt methods to suit the opportunities presented by a child and it's parents, will have most success.
INTRA venous REGIONAL ANAESTHESIA - BIER’S BLOCK

Intravenous regional anaesthesia (IRVA) was first described by Augustus Bier in 1908; his technique was repopularised by Holmes in 1963. The administration of intravenous local anaesthetic in an isolated limb by means of an ischaemic cuff is a simple and effective technique, with a low incidence of failure and high degree of safety.

Clinical Application

IVRA is ideally suited to operations of the distal arm or leg (i.e. below the elbow or knee), such as reduction of a radial or ulna fracture. IVRA is useful for only short surgical procedures; performed in 40 minutes or less (the length of operating time is limited by tourniquet pain, which usually develops after 40 to 60 minutes. IVRA is often a safer option than general anaesthesia, particularly if the patient is elderly, or has cardiovascular or respiratory disease. Of particular importance in hypertensive patients, the tourniquet cuff used must be sealed and inflated to the correct pressure (see below).

Contraindications to IVRA

? Severe Raynaud’s Disease (intermittent arteriolar vasospasm of the distal limbs after cold or emotional stimuli).
? Sickle Cell Disease (IVRA is relatively contraindicated, unless meticulous exsanguination of the limb takes place prior to cuff inflation).
? Crush injury to the limb, IVRA may provoke further tissue damage secondary to hypoxia.
? Age - young children are generally not amenable to IVRA alone, however in combination with sedation and additional analgesia it can be used successfully.
? Patients should be starved, as there may be a possibility of conversion to a general anaesthetic, alternatively the patient may require sedation in addition to IVRA to improve co-operation.

Equipment Required For IVRA

? A single or double tourniquet cuff that has been checked to ensure that it does not leak, and can be inflated 50 to 100mmHg above the patient’s systolic blood pressure.
? Two intravenous cannulae, one for venous cannulation distal to the tourniquet and one for cannulation in the opposite arm to allow access to the circulation if required in the event of complications.
? Full resuscitation equipment and ECG monitoring at all times including immediately after tourniquet deflation.

Drugs Required For IVRA

Prilocaine is the local anaesthetic agent of preference because of its high margin of safety (it has a high therapeutic index). 40ml of 0.5% prilocaine is recommended, although larger volumes will be required for lower limb IVRA (60ml). The maximum dose is 400mg for a 70kg adult (approximately 6mg/kg) which equates to 80ml of 0.5% solution.

Lignocaine is a useful alternative agent. On average 40ml 0.5% lignocaine is required. The maximum dose is 250mg for a 70kg adult (approximately 3mg/kg), which equates to 50ml of a 0.5% solution. Only plain solutions of prilocaine or lignocaine should be used (without adrenaline).

Bupivacaine is unsuitable for IVRA and should never be used due to its cardiotoxic profile (leading to ventricular arrhythmias and death).

IVRA Technique (Figure 1)

? Attach patient to ECG monitor and measure the blood pressure.
? Insert a cannula as distal as possible in the limb to be operated upon.
? Insert a second cannula into the opposite arm for intravenous access (in case of emergency).
? Exsanguinate the limb either with an Esmarch rubber bandage of by simply elevating the limb for several minutes, with brachial / popliteal artery occlusion.
? Protect the upper part of the limb with wadding before placing and inflating the tourniquet to 50 - 100mmHg above their systolic blood pressure (typically 200 to 250mmHg). Check for an absent distal pulse on the limb (radial or dorsalis pedis). During the operation the tourniquet should be observed continuously to check for unintentional slow deflation.
? Inject the local anaesthetic solution slowly via the IV cannula and inform the patient that the limb will feel a little strange and become mottled in appearance. An assistant gripping the forearm during local anaesthetic injection will ensure the most of the anaesthetic solution is retained distally.

? Surgical preparation and draping may proceed about 5 minutes after local anaesthetic injection.

? The tourniquet must remain inflated for a minimum of 20 minutes from the time of local anaesthetic injection.

? Surgical procedures lasting longer than 40 minutes may result in the patient complaining of tourniquet pain, this can be reduced by the use of a double cuffed tourniquet - initially the uppermost cuff is inflated and this can be switched to the lower cuff. The addition of 150mcg clonidine to the local anaesthetic solution may reduce tourniquet discomfort and thus improve conditions. Alternatively, intravenous analgesia such as fentanyl, or ketorolac can be administered (via the emergency IV cannula in the other hand).

? At the end of the procedure the IVRA cannula is removed and the cuff deflated - close observation of the patient is crucial at this point, as this may result in systemic release of local anaesthetic. The patient’s blood pressure should be measured and ECG monitoring continued for at least 10 minutes following cuff deflation.

**Complications**

IVRA is generally a safe technique. The most important complication to recognise is a leaking or accidentally deflated tourniquet cuff - this will result in a large volume of local anaesthetic being rapidly introduced into the circulation. The patient may develop dizziness, nausea, vomiting, tinnitus, perioral tingling, muscle twitching, loss of consciousness, and convulsions. Avoidable deaths have occurred.

**Management of Systemic Toxicity of Local Anaesthetics**

? Airway - Maintain the patient’s airway, administer 100% oxygen and call for help. Turn the patient onto their side; lower their head if possible to prevent aspiration.

? Breathing - start ventilation if breathing inadequate. Intubate if indicated.

? Circulation - pulse check. If in cardiac arrest start CPR. Assistant to start monitoring ECG, pulse oximetry, and blood pressure.

? Conclusions - IV 5mg diazepam or 50mg - 200mg thiopentone. Muscle relaxation if required.

? Hypotension - IV ephedrine 3-6mg increments, elevate legs, IV fluid bolus

**Summary**

IVRA is a simple and effective regional anaesthetic technique to perform, provided that the cuff is checked, and its' pressure monitored.

Resuscitation and monitoring equipment should be readily available when conducting IVRA.
NERVE BLOCKS FOR ANAESTHESIA AND ANALGESIA OF THE LOWER LIMB

Introduction

The purpose of this guide is to provide a detailed, step by step description of how to safely and reliably perform nerve blocks for surgery and pain relief on the lower limb, for the non-specialist anaesthetic practitioner. It covers femoral nerve blockade, lumbar plexus blockade using the inguinal paravascular approach and sciatic nerve blockade. No description is given of more distal blocks, since the majority of the limb is readily anaesthetised with the techniques described and as a group they are relatively simple, reliable and commonly used.

Conduct of nerve blocks

It is recommended that all blocks on major nerves be carried out on patients that are awake or only lightly sedated, as it is believed that this decreased the risk of serious nerve damage, and will not hide the signs of unexpected local anaesthetic toxicity. Sedated patients must still be able to communicate with the operator during the nerve block procedure. It is also recommended that a nerve stimulator be used if one is available as this increases the success rate of the blocks especially in inexperienced hands.

Local anaesthetic drugs and dosages

For fast onset and short duration blocks, lignocaine (maximum 4mg/kg) or lignocaine with adrenaline 1:200,000 (6mg/kg) can be used. When injected into a plexus or near a large nerve such as the sciatic nerve the block will come on at about 10-20 mins and last for 4-8 hours. The adrenaline will prolong the block but may possibly increase the risk of nerve damage through ischaemia. Bupivacaine in a maximum dose of 3mg/kg will give a block of a major nerve that will start at 20-30 minutes and last as long as 18 hours. There is no value in adding adrenaline to bupivacaine except for local skin infiltration.

Anatomy

The nerve supply of the lower limb is derived from the lumbar and sacral plexuses, a network of nerves composed of the anterior primary rami of all the lumbar and the first three sacral nerve roots (and sometimes with a contribution from the twelfth thoracic nerve root). Arising from these plexuses are the five main nerves that innervate the lower limb.

The lumbar plexus: This gives rise to the femoral nerve, obturator nerve and lateral cutaneous nerve of the thigh. The femoral nerve runs in the groove between the psoas major and iliacus muscles, with a covering of these muscles' fascia. It enters the thigh passing under the inguinal ligament, where it is lateral to the femoral artery, whose pulsations are used to help locate the nerve. The femoral nerve block is performed at this point and there are two important features of the anatomy. Firstly, below the inguinal ligament, the femoral nerve divides into anterior and posterior branches, the anterior (superficial) branch supplying sensation to the skin of the anterior and medial thigh and a posterior (deep) branch that supplies the quadriceps muscles, the medial knee joint, and the skin on the medial side of the calf and foot (via the saphenous nerve). Therefore, the block should not be performed lower than just distal to the inguinal ligament, in order not to miss one of the branches. Secondly, as it enters the thigh, the femoral nerve has two fascial layers covering it, the fascia lata and the fascia iliaca. This is in contrast to the femoral artery, which is only covered by the fascia lata alone. This means that the nerve will lie in a different tissue plane than the artery and usually a little deeper. These coverings can be used in blocking the nerve. (See techniques).

The lateral cutaneous nerve of the thigh and the obturator nerve both have important sensory distributions (to the thigh and knee). This article does not cover blocks of these nerves as single entities. However, they can readily be blocked in conjunction with the femoral nerve, using the same technique, to produce a "3-in-1" block and this is described later. (see Blockade of the lumbar plexus using the inguinal paravascular approach).

The sacral plexus: This gives rise to the sciatic nerve and the posterior cutaneous nerve of the thigh. Although these nerves are formed separately within the plexus, they pass through the pelvis and buttock together and with the techniques described here are blocked with the same injection. Hence they are considered here as a single nerve trunk, and unless specifically stated, "sciatic nerve" will refer to both the sciatic nerve and the posterior cutaneous nerve of the thigh.
The sciatic nerve leaves the pelvis and enters the buttock through the greater sciatic foramen, and then passes slightly medial to the midpoint between the greater trochanter and the ischial tuberosity, lying just posterior to the hip joint. It can be blocked at several points along this course (see techniques). The sciatic nerve leaves the buttock, passing out from under the lower border of gluteus maximus muscle and runs distally down the thigh to the popliteal fossa.

**Areas supplied by the individual nerves:**

A pictorial illustration of the skin areas supplied is given in figure 1.

Femoral nerve: supplies skin over the anterior (front) of the thigh, the anterior knee, some of the medial (inner) thigh, via the saphenous nerve it also supplies the antero-medial aspect of the calf down to and including the medial malleolus. It has branches to the hip joint and knee joint and supplies much of the shaft of the femur. Sensation to the medial aspect of the big toe may come from the saphenous (femoral nerve).

Lateral cutaneous nerve of the thigh: supplies sensation to the skin over the lateral (outside) thigh, from the greater trochanter to the knee, and on to the anterior thigh.

Obturator nerve: supplies a small, variable amount of skin on the medial aspect of the knee and lower thigh. More importantly, it has a branch to the knee joint. There is also a small branch to the hip joint.

Posterior cutaneous nerve of the thigh: supplies skin over the posterior (back) thigh, the popliteal fossa, the lower buttock and some of the genital area. Note that this nerve is blocked with the posterior approaches and is often missed with the anterior approach to the sciatic nerve.

Sciatic nerve: via its branches supplies all the skin of the leg below the knee and all the foot, except for the medial calf and ankle, which is supplied by the saphenous (femoral) nerve. The sciatic nerve also has a small branch to the hip joint, a branch to the knee joint and fully innervates the ankle joint.

**Surface anatomy markings**

When it comes to performing the nerve blocks, it is crucial to be able to palpate and accurately locate bony landmarks, since these are the reference points we use for determining the correct site for needle insertion. The following is a description of the bony landmarks used for femoral and sciatic nerve blocks. They are shown in the diagrams of the nerve block techniques.

Anterior superior iliac spine following the iliac crest (ridge of the pelvic bones) from the flanks forwards, it ends in an obvious bony prominence, at the side of the lower abdomen. This is the anterior superior iliac spine.

Pubic tubercle is the bony prominence that can be felt at the inner (medial) end of the groin crease. It is about 2–4 cm from the midline, at the top of the genital area.

Posterior superior iliac spine is the bony prominence at the posterior end of the iliac crest. It is directly caudal to the "sacral dimple"- that depression in the skin visible cranial to (above) the buttocks, on each side, close to the midline.

Greater trochanter this bony landmark is part of the lateral femur, just below the hip joint. It is easy to find at the top of the thigh, protruding directly laterally. With the patient on their side, it represents the highest point on the upper thigh. In obese patients try internally and externally rotating the hip, as this makes the greater trochanter more visible.

The sacral cornu are two bony prominences either side of the midline just at the top end of the natal cleft. One can readily palpate a narrow depression between them - the sacral hiatus. (see figure 3)

The ischial tuberosity is that part of the pelvic bone structure that can be felt posteriorly, on the medial side of the base of the buttock. It is the bony structure that we "sit on."

**Indications for specific nerve blocks**

From the outline of the areas covered by each nerve, the reader should know which blocks would be useful in a given situation. Two points are worth emphasising. The knee joint has significant contributions from femoral, obturator and sciatic nerves and significant injury or surgery to this joint will require that all these be blocked. (For the hip, it is nearly always sufficient to perform a 3-in-1 lumbar plexus block even though there is a small contribution from the sciatic nerve.) Secondly, the area covered by the different nerves may vary considerably and if in doubt, it is best to block both main nerve trunks.

The following are some examples of the possible uses.
Femoral nerve blocks: operations on the anterior thigh, such as repair of large lacerations. Pain relief for fractures of the shaft of the femur, particularly more proximal fractures.

Lumbar plexus (3-in-1) block: all the uses of a femoral nerve block, plus the following: Pain relief and anaesthesia for hip injuries such as dislocations and fractures of the neck of the femur. (Major hip surgery will also require a sciatic nerve block.) Anaesthesia for operations on the lateral thigh such as harvesting of skin grafts, or muscle biopsies. Pain relief for injuries and operations on the knee; extensive injuries and full knee anaesthesia require a sciatic nerve block also this block extends the field of a simple femoral nerve block considerably and is no more difficult to perform.

Sciatic nerve block: pain relief or anaesthesia for injuries or operations on the sole of the foot or any of the toes, such as toe amputation (amputation of the big toe may require supplementation at the medial malleolus as well, because the distribution of the saphenous nerve occasionally extends down the medial side of the big toe). The distribution of the sciatic nerve means that it has fairly limited application as a block on its own and is most often combined with a femoral or 3-in-1 block.

Combined sciatic and femoral or 3-in-1 block: with this combination pain relief and anaesthesia can be provided for almost any injury or operation from the upper thigh downwards. One area sometimes not covered is the upper, inner thigh, and possibly the posterior thigh. This may be a problem with tourniquets applied high on the leg and in this situation some supplementary parenteral analgesia or sedation can be useful. It may be difficult to provide adequate anaesthesia for major hip surgery, although the blocks described will provide good postoperative analgesia. Lancing the dose of local anaesthetic and dealing with possible side effects.

The above discussion will indicate that there are often situations in which one wishes to perform a combined sciatic and 3-in-1 block at the same time. This will necessitate using large volumes of local anaesthetic and the total dose administered may often be at the limit of recommended safe doses. It is important to be able to adjust the concentration of the solution injected when using large volumes, in order to keep the total dose at an acceptable level. (See local anaesthetic, drugs and dosage.)

Local complications of local anaesthetic blocks: The most important is damage to the nerve. Permanent nerve damage is very rare. It may be caused by accidentally injecting local anaesthetic within the nerve itself (intraneural) or by traumatizing the nerve with the needle point. Two signs of intraneural injection are severe pain on attempted injection and marked resistance to injection. (For the patient to respond to the pain of intraneural injection he or she must be awake, or only slightly sedated.) Either of these warning signs should prompt the operator to stop injecting and reposition the needle. Intraneural injection may also be less likely if a short-bevel needle is used. Paraesthesia is the "electric shock-like" feeling felt as the nerve is touched by the needle. It should be a warning sign that nerve damage may occur if the needle is inserted further.

It is also possible to cause a haematoma by puncturing an artery with the needle - most commonly this will be the femoral artery. This is rarely of any significance. If the femoral artery is punctured then firm pressure applied to the site for 5 minutes will minimise the haematoma.

Performing the nerve blocks - patient preparation and techniques. When performing any of the blocks that are described here, the steps taken to safely prepare the patient should be carefully followed.

Preparing the patient
Consent - explain the entire procedure to the patient. This will help to relieve any anxiety and increase co-operation.
Fasting - if an elective procedure is planned, then the patient should be fasted similar to having a general anaesthetic. This increases safety in the event that a general anaesthetic or resuscitation is required.
Monitoring - the potential complications described in the preceding section mean that monitoring is essential. If available, ECG and blood pressure monitoring should be used. If sedation is planned then a pulse oximeter should also be used. In every case, the most useful monitor is to maintain careful, continuous observation of the patient throughout. An assistant can be invaluable in helping with this.
Intravenous access - because of the possible complications, should be intravenous access secured before any block is performed. This also allows administration of intravenous fluids, sedative agents and resuscitation drugs if required.
Positioning - take care with positioning the patient for the block and make sure they are as comfortable as possible as this will make the block easier to perform.
Identify the bony landmarks - these are described in the anatomy section.

Clean the site - the skin over the block site should be cleaned with an antiseptic agent and surrounded with sterile drapes. The operator should wash their hands and wear sterile gloves.

Perform the block!

Allow time for the local anaesthetic to take effect - at least 15 - 20 minutes will be required for surgical anaesthesia. With the weaker concentrations of bupivacaine, 30 - 45 minutes may be required.

Techniques - The femoral nerve and lumbar plexus

Anatomy - The femoral nerve has contributions from the second, third and fourth lumbar nerves. It is derived from the lumbar plexus and in fact lies within the same fascial envelope as the lumbar plexus. This important fact may be utilised to block most of the nerves originating in the lumbar plexus with a single injection distally, as local anaesthetic can be made to spread proximally within this plane. (See anatomy)

Technique (figure 2) The patient lies supine with the leg extended, lying flat on the bed. The operator stands on the side of the patient that is to be blocked. Firstly, identify the point of injection, using the surface landmarks.

For the femoral nerve, this is just below (distal to) the inguinal ligament. Palpate both the anterior superior iliac spine and the pubic tubercle. The line between these two overlies the inguinal ligament. It is often helpful to draw the lines that are described on the skin. The femoral artery should lie at the midpoint of the inguinal ligament and it is necessary to locate this by feeling for the pulse at this point. The site for injection is 1cm lateral to (outside of) the pulsations of the femoral artery and 1 - 2cm below (distal to) the line of the inguinal ligament. Having identified the site, it will be more comfortable for the patient if a small amount of local anaesthetic is used to create a skin wheal ("bleb") at the injection point.

An ordinary needle of length 3 - 4cm and 21 - 23g in width is suitable for performing this block. It should be inserted perpendicular to the skin, but aiming slightly towards the head of the patient. The following are two ways of carrying out the block. In the first technique, the operator attempts to locate the nerve by eliciting paraesthesiae, or failing this by depositing the local anaesthetic over a range of areas (the classical technique of Labat 5). In the second technique, use is made of the fascial layers overlying the nerve and a single injection only is employed (Khoo and Brown2). In both cases, it is very important to remember the anatomy. The femoral nerve lies adjacent to but slightly deeper than the structures contained within the femoral sheath (the artery, vein and femoral canal). This is because the nerve lies deep to the fascia iliaca, while the contents of the femoral sheath lie on top of it.

The classical approach The needle is advanced through the skin, as described above, until the patient feels paraesthesiae in the distribution of the femoral nerve. If a depth of 4 - 5cm is reached and no paraesthesiae are found, then it should be withdrawn to just below the skin and advanced again in a slightly medial or lateral direction, repeating this until the patient feels paraesthesiae. Once this occurs, the needle should be fixed in position with one hand, resting this hand on the patient to try and minimise movement. With the other hand, the syringe containing local anaesthetic is then connected to the needle and gentle aspiration performed. If no blood is seen then 15 - 20 mls of local anaesthetic is injected (aspirating again regularly to check for the presence of blood).

The presence of paraesthesiae is the best indicator for correct positioning of the tip of the needle, but it is often not easy or even possible to locate the femoral nerve in this manner.

Alternatively as the needle is advanced alongside the artery, its pulsations may cause lateral (side to side) movement of the hub of the needle. If this is the case, the needle is slowly advanced and frequent observations made until the point is reached when the later movements are at their greatest. This generally represents a depth where the tip of the needle is just deep to the artery and should be in the correct plane. The needle is then fixed, the syringe connected and aspiration performed as before. However, only 10 mls of local anaesthetic is injected at this site. This injection is then supplemented by withdrawing the needle slightly redirecting the tip outwards (laterally), inserting to the same depth as before and injecting 3 - 4 mls of local anaesthetic (after aspiration). This process is repeated 2 or 3 times, moving progressively further laterally, such that a total of 20 - 25 mls of local anaesthetic is deposited in a "fan-shaped" area lateral and deep to the femoral artery.

The single injection technique2 This method has the virtue of simplicity and generally involves less probing with the needle. For this reason it is popular but requires some practice for a high success rate.

The site for injection is the same as already described. However, the needle is inserted directly perpendicular to the skin. If the needle is held gently between thumb and forefinger, then a slight resistance is encountered at the
fascia lata, followed by a definite loss of resistance, or "pop" as the needle penetrates this layer. The same thing is felt as the needle penetrates the fascia iliaca and comes into the proximity of the femoral nerve. Therefore, immediately on feeling this second loss of resistance, or "pop", the tip of the needle should be in the correct position. The needle is then fixed in position with one hand, the other hand again being used to connect a syringe, aspirate to check for blood and inject 20 ml of local anaesthetic.

This technique is entirely dependent on being able to detect the two points of loss of resistance as each of the fascial layers is penetrated. This is much easier if a short-bevel needle is available to use, as it does not pierce the fascia quite as easily as an ordinary needle, making the feel of the layers more obvious. If a short-bevel needle is not available, then the same effect can be achieved using an ordinary needle but blunting it prior to insertion. The "blunting" may be cleanly achieved by piercing the side of the protective plastic sheath (that comes with the needle) several times with the needle tip before performing the block. The detection of paraesthesiae is not to be recommended when using this technique as the "blunted" needle is more likely to cause nerve damage.

Use of a nerve stimulator If an insulated stimulating needle is available for use, then it is necessary to obtain contraction of the quadriceps muscle group. This is most reliably seen by movement of the patella and extension of the knee joint. (The movement of the knee is not normally obvious, as the patient's leg is usually flat on the bed and fully extended anyway.) The contractions should still be visible at a stimulating current of 0.3 - 0.5 mA indicating adequate proximity to the nerve. Exactly the same technique will be used if one wishes to perform a lumbar plexus block using this approach.

**Blockade of the lumbar plexus using the inguinal paravascular approach**

This technique is also referred to as the "Winnie 3-in-1 block" after the author who first described it. It is so called, because it aims to block three nerves with the one injection: the femoral nerve, the lateral cutaneous nerve of the thigh and the obturator nerve.

**Anatomy** - For most operations on the thigh and knee it is not sufficient to block the femoral nerve alone. The lateral cutaneous nerve of the thigh supplies all the outside of the thigh and the obturator nerve supplies a variable amount of skin on the inner thigh just above the knee and contributes to the innervation of the knee joint.

All three nerves are derived from the lumbar plexus. The plexus lies on the quadratus lumborum muscle and behind the psoas major muscle and is invested in a fascial sheath derived from these two muscles. This sheath forms a continuous covering around the plexus, extending down to the femoral nerve just below the inguinal ligament. Therefore if local anaesthetic injected around the femoral nerve at the inguinal ligament can be made to spread proximally, then the other two nerves can be simultaneously blocked at their origins from the lumbar plexus.

**Technique** - The injection point and method for correct needle placement are exactly as described for the femoral nerve. It is most practical to use the single injection technique, as this will enable placement of the large volumes required.

There are two differences; the main difference comes with the actual injection of the local anaesthetic. Having aspirated on the needle to check that the tip is not intravascular, the hand is then moved to apply firm pressure on the thigh (with the thumb) about 2 - 4 cm. below the insertion point of the needle. The injection is then performed, all the while maintaining the pressure. The pressure can be released about thirty seconds after the injection has been completed. (The injection will require an assistant, as the operator will have one hand immobilising the needle and the other applying pressure.) This procedure encourages spread of the local anaesthetic upwards, towards the lumbar nerve roots.

The second difference is that larger volumes of local anaesthetic are used to achieve the necessary spread. The minimum volume to block all three nerves is 20 mls. However, many texts suggest larger volumes such as 25 – 30 mls. When using these volumes, particularly in combination with a sciatic nerve block, it may be necessary to dilute the concentration of local anaesthetic solution used in order to limit the total dose given.

**Use of a nerve stimulator:** as for the femoral nerve.

**The Sciatic Nerve**

**Anatomy** - The sciatic nerve is the largest nerve in the body, measuring about 2 centimetres in thickness in its proximal portion. In this portion it is actually made up of the sciatic nerve and the posterior cutaneous nerve of the thigh. This "double nerve" contains contributions from lumbar nerve roots 4 and 5 and sacral nerve roots 1,2 and 3. In the techniques that are described here, this large "double nerve" is considered effectively as a single nerve and blocked with the one injection. For simplicity, it is referred to just as the sciatic nerve.
The important bony landmarks that one needs to be able to identify for blocking the sciatic nerve via the two posterior approaches described are the greater trochanter, posterior superior iliac spine, the ischial tuberosity and the sacral hiatus. For the anterior approach, the landmarks are the anterior superior iliac spine and the pubic symphysis on the pelvis and the greater trochanter on the femur.

Technique - It will be appreciated from the description of these landmarks that there are several possible routes to block the sciatic nerve. Three of the most common approaches are described here. The first is the classical posterior approach of Labat performed with the patient in the lateral position. The second is another posterior approach, but the patient is supine and the leg is flexed at the hip and at the knee. Finally, the anterior approach is described where the patient is supine with the legs lying naturally extended. The choice of technique will to some extent be influenced by the position that is easiest for the patient to assume. However, the success rate is higher with the posterior approaches unless a nerve stimulator is used. Furthermore, the anterior technique tends to be technically more difficult and therefore it is suggested that every effort should be made to position the patient for one of the posterior approaches.

Posterior approach of Labat (figure 3) The patient is first placed in the lateral position with the side to be blocked uppermost. While the lower leg is kept straight, the upper leg is flexed at the knee so that the ankle is brought over the knee of the lower leg. Another way of achieving the correct degree of hip and knee flexion is to have the posterior superior iliac spine, the greater trochanter and the knee in a straight line. The point of injection is identified as follows:

A line is drawn between the greater trochanter and the posterior superior iliac spine (the line lies approximately over the upper border of the piriformis muscle). From the midpoint of this line, at right angles to it, draw a second line passing down over the buttock. The point of injection is 3 - 5 centimetres along this perpendicular line. It can be more precisely identified by drawing a third line between the greater trochanter and sacral hiatus, the point of injection being where this third line intersects with the second, perpendicular line. Having identified this point, place a small wheal of local anaesthetic at the site.

The needle that is required for this block needs to be quite long. A standard adult lumbar puncture needle is usually sufficient (9cm, 22G). In a very large person, an extra long needle, (10 - 12cm) may make the location of the nerve an easier task.

The needle is inserted perpendicular to the skin and slowly advanced until either bone is encountered, or paraesthesiae are elicited. (For the block to be successful, paraesthesiae below the knee should be felt.) If bone is encountered, the needle is withdrawn approximately 1-3cm and redirected slightly, either medially or laterally. Gentle probing within this single (transverse) plane should enable the nerve to be located by producing paraesthesiae as described. If the needle has been inserted as far as possible and neither paraesthesiae elicited nor bone encountered then the tip may have entered the greater sciatic notch. Should this occur, then the needle should be withdrawn almost fully, until the tip is just beneath the skin and then redirected in a slightly medial or lateral plane as described above.

Having located the nerve by paraesthesiae, the needle should be fixed in position and a syringe containing approximately 20ml of local anaesthetic connected. Aspiration is performed to exclude intravascular placement of the needle and the local anaesthetic is then injected. Repeat aspiration half way through the injection, in case the tip of the needle has moved). It is important that if severe pain occurs or if there is significant resistance to injection then the operator should stop immediately and reposition the needle, as these may be signs of intraneural injection.

Alternative posterior approach (of Raj) If it is not possible to have the patient lying on their side, then a variation of the posterior approach may be performed with the patient supine, although the hip is still manipulated.

To perform this block, the operator stands by the patient's bed, on the side to be blocked. The hip is then flexed as much as possible with knee bent. This position can be held stable by bracing the foot against the front of the operator's shoulder as they face towards the head of the bed. Alternatively, an assistant can hold the leg steady. The greater trochanter is palpated on the outside of the leg and the ischial tuberosity is also located, being the main prominence at the base of the buttock. It is often possible to palpate these two bony protuberances at the same time using the thumb and middle finger of the same hand. The midpoint between these two landmarks is the injection point.

It is sometimes possible to identify this line joining the greater trochanter and ischial tuberosity as a depression between two muscle bellies - semitendinosus and biceps femoris.
Having located the injection point a small skin wheal is raised and the same needle as above is inserted at right angles to the skin. Once again the aim is to elicit paraesthesiae below the knee and this is achieved exactly as described above - by gentle probing in the transverse ("side to side") plane. The sciatic nerve is most likely to lie slightly medial to the path of the needle if these landmarks are used. The injection of local anaesthetic is then also performed in exactly the same manner as for the classical technique of Labat.

It should be noted that this alternative approach would block the sciatic nerve several centimetres more distally than the first approach described. However, it is rare to miss the posterior cutaneous nerve of the thigh, even with the alternative approach.

Anterior approach to the sciatic nerve Occasionally, it will be not be possible to move the patient's leg from the neutral position with them lying supine. In this case a sciatic nerve block can be performed using an anterior approach although as already stated, this tends to be more of a technical challenge! This technique will also result in the sciatic nerve being blocked at a relatively distal point (just beyond the hip joint) and hence it is possible to miss the posterior cutaneous nerve of the thigh, which becomes separated from the sciatic nerve by the hamstring muscles just below the buttock.

The landmarks for the point of injection are as follows: (see figure 4) firstly, trace a line over the inguinal ligament (from the anterior superior iliac spine to the pubic tubercle) and divide it into thirds. From the junction of the inner and middle thirds draw another line at right angles to the first, going down the leg. The next step is to find the greater trochanter and from this draw another line parallel to the line over the inguinal ligament (the first line drawn). Where this last line crosses the perpendicular line (the second line) is the point of injection. A small skin wheal of local anaesthetic is then injected at this site.

The needle used for this block is a standard adult lumbar puncture needle (9 cm long). However, the depth of insertion required for this block is commonly greater than for the posterior approaches and a longer needle is often required.

The needle is then inserted perpendicular to the skin, which means aiming in slightly lateral direction. The operator should aim to strike bone, close to the medial edge of the femur. This will be at about the level of the lesser trochanter. The needle is then withdrawn slightly, redirected more medially (it should be more towards the vertical) and advanced, this being repeated until the needle is "walked off" the bone, the aim being to just slip past the medial edge of the femur. The operator should note the depth at which the needle initially strikes the femur. Normally this is done by carefully observing the portion of needle shaft remaining at the skin. Once the needle has been directed off the bone, as described above, it should be inserted a further 5 cm (2 inches). The tip of the needle should now be in the region of the neuromuscular bundle. (See figure 5)

Aspiration to check for intravascular needle placement is particularly important if this approach is used, as there is a higher likelihood of vascular puncture. Having aspirated, the needle is immobilised in the usual fashion and approximately 20 ml of local anaesthetic injected.

If there is resistance to injection, then the tip of the needle may still be within muscle substance. If this occurs then the needle should be slowly advanced until injection is easily accomplished.

This technique does not require that paraesthesiae are found, but if they are elicited this is a positive sign of correct needle placement.

If one wishes to be positive about the needle placement and it is not possible to elicit paraesthesiae using the anterior approach as just described, then it is sometimes helpful to use a more medial injection point (about 1 - 2cm inside of the one described). This means that the needle will pass the medial edge of the femur at more of an angle than before and the tip will end posterior to the femur. This may help find the nerve, which tends to lie slightly behind the femur at this level. (When using this more medial injection point, it may help to place the free hand under the buttock and palpate the ischial tuberosity. The needle is then aimed at a point estimated to be 1 - 2cm lateral to the ischial tuberosity.)

Performing a sciatic nerve block using a nerve stimulator A nerve stimulator may be used in conjunction with any of the approaches to the sciatic nerve that have been described above. The techniques for determining the point of injection and locating the nerve are no different, except that one will look for muscle contraction. The best indicator of proximity to the nerve is dorsiflexion of the foot at the ankle and one should aim to achieve this at a stimulating current of 0.3 - 0.5 mA. However, when using the posterior approaches, one may also see contraction of the "hamstring" muscles down the back of the thigh, which may be taken as a sign of proximity to the sciatic nerve.
Having achieved muscle contraction at the required stimulating current, injection of local anaesthetic is performed in the usual manner.
POSTOPERATIVE ANALGESIA IN PAEDIATRIC DAY CASE SURGERY

Introduction

Paediatric day case surgery was first described in 1909 by James Nicoll, who performed 8988 operations as day case at the Royal Glasgow Hospital. Since then, day case surgery has continued to grow and now about 50% to 60% of paediatric surgery is performed as outpatients in most of the western countries like USA and UK. In India, the incidence of paediatric day case surgery is low, i.e., 35%. This is because of illiteracy, lack of proper transport facilities and unhygienic conditions at home.

Key to success in paediatric day case surgery is proper selection of patients, prevention of common postoperative complications and adequate pain management. Severe postoperative pain not only decreases the patients' functional capacity but also is associated with longer postoperative stay and higher incidence of unanticipated readmission. Pain may precipitate postoperative nausea vomiting (PONV) which is another cause of unanticipated readmission. Hence adequate pain management is mandatory in day case surgery.

Planning for postoperative analgesia must be done during the preoperative visit, keeping in mind the age, psychological and ASA status of the patient, and the type of surgery. Appropriate assessment of pain is essential for providing optimal analgesia.

Assessment

Numerous scoring systems are available for assessment of pain in paediatric patients. Each system has its own advantages and disadvantages. Selection of scoring systems mainly depends upon the age of the child.

Neonates. Day case surgery is not contraindicated in full term neonates - minor procedures like examination under anaesthesia and incision and drainage can be performed. Fortunately, these procedures do not produce much postoperative pain.

A variety of assessment tools have been developed for neonates. Observation of facial expression, body position and movement, crying, arterial pressure, heart rate, skin colour, ventilatory frequency and sleeplessness are used to find out the severity of pain in neonates. But these parameters can be altered by non-painful stimuli. Therefore a more rational approach is to assess the improvement of behavioural or physiological parameters in response to comfort, analgesia or sedation.

Infants and Children up to 3 years. Like neonates, assessment of pain in this age group of children is also based on behavioural and physiological response to comfort and analgesic therapy. Though exhibited behaviour may be more vigorous with an "all or nothing" type of response, sometimes the response is more precise and they can locate the pain. Objective pain scale (OPS) and toddler-preschooler postoperative pain scale (TPPPS) are commonly used to assess the intensity of pain.

Children aged 3 to 7 years. These patients can differentiate the presence or absence of pain and locate the pain. They can also express the intensity of pain in the form nil, mild, moderate and severe. The face scale or Oucher scale can be used in this age group. Children of five or more years old can operate visual or colour analogue scales for expression of pain.

Older Children. Like adults, children more than seven years old can express intensity, location and quality of pain. Any scoring system such as horizontal VAS, vertical colour analogue scale and self reporting are effective and reliable.

Management of Postoperative Pain

Operative procedures associated with severe postoperative pain should not be performed as day surgery. For most patients postoperative pain should not be a major problem provided that local anaesthesia and NSAID have been used either as a part of the anaesthetic technique or after completion of surgery. Oral analgesics are the mainstay of pain relief at home.

Topical Anaesthesia. EMLA cream is an eutectic mixture of prilocaine and lignocaine and is very effective at providing dermal anaesthesia. Topical EMLA decreases the pain associated with circumcision, release of preputial adhesion, myringotomies and skin grafting. To obtain effective analgesia cream should be applied to the skin with an occlusive dressing about 45 to 60 minutes prior to surgery. Duration of analgesia is about 1 st hours. EMLA should be used with caution in infants less than 3 months of age or in patients who are taking sulphonamides or other methaemoglobin inducing medications because of potential of methaemoglobinidaemia.
Lignocaine gel can be used to provide analgesia following circumcision and after repairs of lacerations. Parents can be taught to apply the gel for postoperative analgesia during first 24-36 hours. Application of bupivacaine and epinephrine (adrenaline) on the open wound towards the end of surgery provides excellent analgesia. Topical local anaesthetic eye drops can be used to provide analgesia following ophthalmic surgery.

Instillation. Bupivacaine instillation before closure of small wounds is very effective. Continuous infusion of 0.25% bupivacaine through a small cannula at a rate of 1-3ml/hour provides a simple, safe and effective method of analgesia at the donor site of skin graft or iliac crest bone graft.

Wound infiltration. Local anaesthetic agents may be administered intradermally or subcutaneously to block impulse conduction in local nerve fibres. Surgical wound infiltration can be used to provide analgesia following skin biopsies, muscle biopsies and virtually all procedures where other regional blocks are either inappropriate or contraindicated.

Caudal epidural block is widely used in paediatric patients to provide analgesia following surgery below the level of the umbilicus. With a single injection, it provides long lasting postoperative analgesia in paediatric day case surgery.

Caudal block is achieved by injecting local anaesthetic agents into the epidural space through the sacral hiatus, which is situated 1 to 2cm above the gluteal crease, superior to the coccyx and between the prominent sacral cornuae. The sacral hiatus can be located by drawing an equilateral triangle of which the two superior angles overlie the posterior superior iliac spines and third angle overlies the sacral hiatus (see Update in Anaesthesia No. 9 1998).

Under general anaesthesia the patient is placed in the lateral position. The skin is prepared using a standard sterile technique. The block is performed using a short bevelled needle of less than 3cm length to reduce the incidence of accidental dural puncture. The needle is inserted through the sacral hiatus at a 45 degree angle pointing rostrally (towards the head). Once the sacroccocygeal ligament is punctured the angle of the needle is decreased to 20 degrees. Approximately 0.75 to 1ml/kg of local anaesthetic agent is required for analgesia up to T10 level.

Weakness of the lower limbs associated with caudal block may delay the discharge of the patient. This can be minimised by using weaker local anaesthetic solutions such as 0.125% bupivacaine. Another drawback of single shot caudal block is its short duration. The duration can be prolonged by adding drugs such as clonidine an a2 agonist, in a dose of 1-2mcg/kg or preservative free ketamine in a dose of 0.5mg/kg. Morphine and other spinal opioids are not recommended for paediatric day case surgery because of the risk of delayed respiratory depression.

Peripheral Nerve Block. Peripheral nerve blocks such as penile block, inguinal block, fascia iliaca block and sciatic nerve block have been demonstrated to be as effective as single shot caudal block. Moreover they produce longer lasting analgesia.

Penile block is performed to provide analgesia following circumcision, minor hypospadius surgery and other distal penile procedures. Different techniques have been described to block penile nerves including a midline and paramedian approaches. The paramedican approach is often preferred due to a lower incidence of complications such as intravascular injection, haematome and ischaemia. A short bevelled needle is inserted perpendicular to the skin at the inferior edge of the symphysis pubis at the 11 and 1o'clock positions. The needle is advanced until Bucks fascia is penetrated, which is determined by a loss of resistance. After careful aspiration plain 0.5% bupivacaine 1ml + 0.1ml/kg is administered. For better effect, subcutaneous infiltration of local anaesthetics at the base of the penis from 3 to 9o'clock position is recommended. However a full ring block should be avoided. Ilioinguinal and iliohypogastric blocks provide effective analgesia after inguinal herniotomy and orchidopexy. The quality and duration of analgesia achieved by this block are comparable to caudal block.

A short bevelled 22 to 25 gauge needle is inserted, one patient's finger breadth medial to anterior superior iliac spine. After penetrating the external oblique aponeurosis and the internal oblique muscle fascia, a sudden loss of resistance is felt and the local anaesthetic can be deposited after a negative aspiration test. A dosage of 0.4 ml/kg of 0.25% bupivacaine with or without adrenaline is used for unilateral ilioinguinal and iliohypogastric nerve blocks.

Another injection immediately lateral to pubic tubercle to block the nerves coming from the opposite side and local infiltration along the incision improve the quality of analgesia.

In about 50% patients the subcostal nerve accompanies the iliohypogastric nerve and may be responsible for inadequate pain relief. Therefore a more effective block can be achieved by an injection directed laterally to contact the inside wall of the ilium and infiltrating local anaesthetic as the needle is withdrawn slowly. For pain
relief after orchidopexy ilioinguinal and iliohypogastric blocks must be combined with local infiltration of the scrotum. This is because the inferior aspect of the scrotum is innervated by the pudendal nerve. Brachial plexus block may be used to provide postoperative analgesia following upper extremity surgery. The axillary approach is the safest, more reliable and most commonly used in children and may provide useful analgesia for operations below the elbow.

Positioning of the patient is very important to make the artery (which is surrounded by the nerve plexus) palpable. The child is placed supine and the arm is abducted to 90 degrees and rotated externally. The forearm is flexed to 90 degrees. A short bevelled needle is inserted perpendicular to skin at the most proximal part at which the artery can be palpated. The needle is advanced until a "fascial click" is felt. At this point arterial pulsation is usually transmitted to needle. These two signs indicate that the needle tip is within the fascial sheath. After a negative aspiration test, local anaesthetic agent may be injected. Bupivacaine 0.25%, 0.6ml/kg is usually adequate. A two point injection technique, i.e., one above and another below the artery improves the success rate.

Femoral nerve block and 3 in 1 blocks are indicated in day surgery to provide analgesia following skin grafting where the graft is taken from thigh and muscle biopsies. However due to the effect on the leg muscles, postoperative mobilisation is significantly affected which may delay discharge.

The femoral nerve is situated just lateral to the femoral artery below the inguinal ligament deep to the fascia lata and iliac. Therefore when the needle is advanced, 2 losses of resistance must be felt. Usually 0.25% bupivacaine 0.3ml/kg is enough for adequate blocks of the femoral nerve (See Update in Anaesthesia No. 11 2000). In a 3 in 1 block apart from the femoral nerve, the lateral cutaneous nerve of thigh and obturator nerve are also blocked. The volume of local anaesthetic should be doubled so that it can spread adequately between the iliacus fascia and muscle to reach the other nerves. Distal pressure on the femoral sheath during and after the injection improves the quality of nerve block.

Greater auricular nerve block. This nerve innervates most of the pinna and may be blocked to provide excellent analgesia after otoplasty. The block is performed by injecting 0.5% bupivacaine 1ml subcutaneously between the mastoid process and the descending ramus of the mandible.

Systemic Analgesics

Non-Steroidal Anti-Inflammatory Drugs (NSAIDs) along with local anaesthesia are the mainstay of postoperative pain relief in paediatric day case surgery. They have several advantages over opioid analgesics including a lack of respiratory depression and sedation. They do not cause nausea or vomiting.

NSAIDs have been found to be very effective analgesics in older children. However use of these agents are not recommended below one year of age due to the possibility of immature renal function and hepatic metabolism. Diclofenac, ibuprofen and ketorolac are the most commonly used agents. Administration of these agents before surgery as a premedicant provides optimal analgesia due to their anti-inflammatory activity.

Bronchospasm induced by NSAIDs is very rare in children and asthma is not a contraindication to the use of NSAIDs. However one should avoid them if the child has been recently or repeatedly hospitalised with asthma, or has required steroids systemically, or is known to be NSAID sensitive (Table 1).

Paracetamol (acetominophen) is a very safe and effective analgesic in children including infants and neonates. Oral paracetamol 20mg/kg as a premedication is useful in achieving therapeutic plasma concentration postoperatively. The total daily dose of paracetamol can be up to 90mg/kg/day for the first 3 days in healthy children. This should be reduced to 60mg/kg/day in neonates (Table 2). The drug may be administered rectally but higher doses are necessary, due to poor and erratic absorption through the rectal mucosa.

Opioids are not ideal for paediatric day case surgery as they may produce ventilatory depression, excessive sedation and postoperative nausea and vomiting. With some procedures however opioids are required during and after surgery to control pain. Shorter acting opioids are ideal - fentanyl (1-2mcg/kg) is commonly used. Longer acting opioids (morphine / pethidine) may be required if postoperative pain is unexpectedly severe. Although the procedure may have been planned on a day case basis unexpected hospital admission may be required for control of severe pain.

Non Pharmacological Therapy may be helpful in some children. It includes distraction techniques like playing with toys, watching videos, music and hypnotic therapy. The child may be allowed to stay in a friendly atmosphere preferably with parents in the immediate postoperative period. All these measures reduce analgesic requirement and speeds recovery.
Conclusion
Postoperative pain following day case surgery in paediatric patients is usually not severe and diminishes within 3 to 5 days. Peripheral nerve blocks by local anaesthetic agents provide optimal analgesia in the immediate postoperative period. Patients should not be discharged until pain is well controlled with oral medications such as paracetamol, ibuprofen or diclofenac.
THE PHARMACOLOGICAL MANAGEMENT OF NEUROPATHIC PAIN

Introduction

Tissue injury is usually accompanied by pain and is described as neuropathic if the initiating injury occurs to neural tissue. After injury occurs, symptoms are initially experienced distal to the site of injury: by contrast in non-neuropathic pain (nociceptive pain) symptoms are apparent, at least initially, at the site of injury. With time the margins between these types become blurred and each may coexist with the other. The consequence of neural injury is change in neural function both at the site of injury and proximal to it with the symptoms produced being manifestations of neural over or under activity. Typical features of neuropathic pain, regardless of the causal injury, include shooting / lancinating pain, burning pain, paraesthesia / dyseaesthesia, numbness and allodynia (pain produced by a normally non-painful stimulus).

In addition to differing symptoms experienced with neuropathic and nociceptive pain, there are differences in those therapeutic agents which can produce pain relief. For example, it is accepted that nociceptive pain may be relieved by morphine and non-steroidal anti-inflammatory drugs (NSAIDs). However, with neuropathic pain some studies suggest analgesia with morphine (1,2) and NSAIDs (3,4), while others demonstrate no analgesia with morphine (5,6) or NSAIDs (7,8).

The aim of this article is to highlight current therapeutic options for the treatment of neuropathic pain.

Capsaicin

It has been recognised for almost 150 years that the topical application of extracts of the capsicum pepper can produce pain relief (9). It is now recognised that the active pain killing constituent of the chili pepper is capsaicin, which when repeatedly applied topically in appropriate concentration causes reversible depletion of the neurotransmitter substance P (SP) from the sensory nerve endings (10) and hence pain relief, which may take several weeks to occur. Topical application of capsaicin has been shown to reduce the pain of a variety of conditions, including post herpetic neuralgia (11 - 13), painful diabetic neuropathy (14 - 16), chronic distal painful polyneuropathy (17), surgical neuropathic pain (18), post mastectomy syndrome (19) and osteoarthritis (20 - 23). The major side effect is that of burning discomfort which may lead to poor patient compliance. The addition of glyceryl trinitrate (GTN) to capsaicin reduces the burning discomfort associated with application (24, 25) and may improve compliance. GTN is also known to have an anti-inflammatory effect (26, 27) and this may augment the analgesia from the capsaicin.

Tricyclic antidepressants

It is widely accepted that oral tricyclic antidepressants (TCAs) have an analgesic effect in neuropathic pain (28, 29) with evidence of efficacy existing for amitriptyline (30 - 34), imipramine (35), desipramine (36 - 38) and clomipramine (39, 40). This analgesic effect is independent of their antidepressant effect (41) and may be dose related (42,43). TCAs have an effect of 5 hydroxytriptamine release (44), the noradrenergic pathways (45) and a sodium channel blocking effect (46), the later effect being shared by the local anaesthetic and anticonvulsant groups.

Unfortunately, the undoubted analgesic effect of the TCAs is tempered by their side effect profiles with somnolence and dry mouth being the predominant side effects. Recent work has highlighted a potential analgesic effect of topical doxepin, a TCA, in neuropathic pain (47,48). The topical application of doxepin is associated with few side effects, and particularly central side effects. Animal work has suggested a potential peripheral action of TCAs (49, 50) and may explain the analgesia seen in human studies.

Anticonvulsants

It has long been appreciated that there are similarities between epilepsy and neuropathic pain (in 1885 Trousseau described trigeminal neuralgia as "epileptiform neuralgia 51") and that drugs that are effective in reducing seizure frequency may also have an analgesic effect in neuropathic pain (52, 53). The first report of analgesia with an anticonvulsant in neuropathic pain was with phenytoin in 1942 (54). Subsequent randomized controlled trials (RCTs) has confirmed this case report evidence with phenytoin (55, 56). In 1962 case report evidence of analgesia with carbamazepine emerged (57), with subsequent support from RCTs in trigeminal neuralgia (58) and painful diabetic neuropathy (59). Carbamazepine remains the most frequently used anticonvulsant for neuropathic pain (60).

Anecdotal evidence points to a similar analgesic effect with lamotrigine (61 - 64), although the evidence from the small number of RCTs so far reported is mixed (65, 66). The analgesia from lamotrigine may be dose related and studies reporting no analgesia used low dosing regimes of this drug.
Gabapentin, a structural analogue of the inhibitory neurotransmitter gamma amino butyric acid (GABA), which paradoxically is thought not to exert its effect on GABA receptors (67), has recently been demonstrated to reduce neuropathic pain (68-73), and in particular post herpetic neuralgia (74) and painful diabetic neuropathy (75), both conditions being archetypal neuropathic pain conditions. The potential advantage of lamotrigine and gabapentin over carbamazepine are their more favourable side effect profiles (76).

When oral dose titration is not possible then parenteral administration may be necessary. Intramuscular fosphenytoin (a water soluble ester pro-drug of phenytoin, lacking its infusion related side-effects) produces analgesia where neuropathic pain is present (81). Intravenous infusions of phenytoin and fosphenytoin both have a similar analgesic effect with the added advantage of relief that extends beyond the period of infusion and the intravascular half-life of the drug (82, 83).

Despite a common effect (reduction in seizure frequency and analgesia), anticonvulsants differ in their mode of action. Phenytoin has a sodium channel blocking effect (77) while lamotrigine has an effect on voltage gated cation channels (78) and glutamate release (79), while gabapentin appears to exert its action via the alpha delta 2 sub unit of the calcium channel (80). The clinical consequence of these differing modes of action is that a failed trial with one anticonvulsant does not mean that another in this class will not work.

Baclofen

Baclofen, like gabapentin, is structurally similar to the inhibitory amino acid gamma amino butyric acid (GABA) and yet seems to have a mechanism of action that differs to that of GABA (84, 85). It is known to depress release of the excitatory neurotransmitters glutamate and aspartate (86, 87). Isolated studies suggest an analgesic effect in trigeminal neuralgia (88, 89).

CCK Antagonists

The causes of incomplete analgesia with opiates in neuropathic pain are many. Among them are elevation of the anti-opioid peptide cholecystokinin (CCK) (90-92). Neural injury produces an elevation in plasma CCK levels (93), and if a CCK antagonist is administered, then opiate sensitivity in neuropathic pain may return. The obsolete anti-ulcer drug proglumide is a non-specific CCK antagonist (94) and has been shown to augment the analgesic effect of sustained release morphine in neuropathic pain (95, 96). As well as reducing the analgesic effect of opiates, CCK is also elevated with chronic opiate administration, and hence more opiate is required to achieve the same level of analgesia with the passage of time (tolerance) (97-103). CCK antagonists such as proglumide can reverse this tolerance. It may therefore be that opiates can be used to treat chronic neuropathic pain when co-administered with a CCK antagonist, although the debate will continue as to whether a dose limit be put on the sustained release morphine preparation.

Membrane Stabilisers

For many years intravenous infusions of local anaesthetics have been used in the management of both acute and chronic pain: the analgesia of IV novocaine was described in 1943 (104). Despite much anecdotal evidence of analgesia with IV lignocaine (105-8) there are few RCTs to verify this effect. It does seem that a short term infusion (e.g. 24 hours) may give relief in some patients for a sustained period (weeks to months).

Parenteral local anaesthetics seem to suppress the activity of spontaneously active fibres in neuromas (109), depresses C - afferent fibre evoked activity in the spinal cord (110) and silence dorsal root ganglion discharge without blocking nerve conduction (111).

Parenteral administration of local anaesthetics may not always be feasible and there is some evidence to suggest that the oral equivalent mexiletene may also have an analgesic effect (112).

Ketamine

Ketamine, a N-methyl D-aspartate (NMDA) receptor antagonist can have an analgesic effect in neuropathic pain (113-4). Its use is associated with side-effects that limit its use, but recent work has suggested an opiate potentiating effect that may be apparent at otherwise sub-therapeutic doses (115).

Nerve blocks

The perineural injection of drugs seems to produce pain relief in some patients with neuropathic pain for varying lengths of time. Local anaesthetic injection may give only short term relief. However, the addition of corticosteroid may lengthen the pain relief produced. Steroids reduce inflammation by reducing prostaglandin synthesis (116), suppress ectopic discharge (117), have C fibre membrane stabilising effects (118) and stabilise the dorsal horn cell (119) and any or all of these effects may contribute to the pain relief produced. With the example of epidural
steroid administration there is mixed evidence with some reports claiming benefit (120), while others suggest less significant benefit in terms of extent and duration of relief (121 - 2)

Clonidine
The alpha adrenoreceptor agonist clonidine has been used for many years as an antihypertensive agent. When administered by the epidural or intrathecal route it has an effect on the descending noradrenergic pathways (123 - 4) which produces analgesia (125 - 8). Unfortunately oral administration is not associated with such relief.

Discussion
While many other agents may be used in treating neuropathic pain, their use is not verified by appropriate studies. It is hoped that the rational use of drugs increases the chance of achieving analgesia in the patient with neuropathic pain. However, when one considers the "numbers needed to treat" (N.N.T.), that is the numbers of patients needing to take the drug to achieve 50% reduction of that symptom in one patient, for the medication used in neuropathic pain it is clear that it is always greater than 2.5 (28,52). Consequently, no one therapeutic intervention is guaranteed of success. This is similar to drugs used in nociceptive pain where the N.N.T. varies from 3.1 with paracetamol 500mg / codeine 60mg, to 3.6 with paracetamol 600 or 500mg, to 9.1 with codeine 60mg (129). Consequently it may often be necessary to work ones way through a list of treatment options before analgesia is achieved. Inevitably any relief produced may be tempered by the associated side-effects of that drug so that improvement in quality of life (pain reduction, mood elevation, increased mobility, better sleep with minimal side effects from treatment) is the therapeutic goal. Poly pharmacy is a real danger, with patients staying on medication in hope of relief when none is actually apparent. Trials of medication for a defined period of time with assessment before and after may be more appropriate.

Pharmacological management will produce the desired analgesia in some, but not all, patients. In those who fail to respond, other modalities of treatment may be considered, ranging from behaviour modification and fostering of coping skills to the more major invasive medical techniques. It is still reassuring, however, to realize that in the future we have the prospect of additional agents which may or may not prove useful analgesics in neuropathic pain. These include agents with more specific sodium channel blocking effects, calcium channel blockers and new generation anticonvulsants and capitalise on the major expansion in knowledge generated from the work of the basic scientists.

It is hoped that this paper highlights the current outpatient therapeutic options and demonstrates a rational approach to the management of the patient with neuropathic pain.
PAIN RELIEF IN LABOUR

Introduction
Giving birth is a painful process. This applies to all social and ethnic groups and has probably been so since mankind walked upright. It is very difficult to measure pain which is recognised via the signals carried through the nervous system and the woman's intellectual response to the stimulus.

Physiology of pain in labour
Labour pain is the result of many complex interactions, physiological and psychological, excitatory as well as inhibitory. Pain during the first stage of labour is due to distention of the lower uterine segment, mechanical dilatation of the cervix and lastly due to stretching of excitatory nociceptive afferents resulting from the contraction of the uterine muscles. The severity of pain parallels with the duration and intensity of contraction. In the second stage additional factors, such as traction and pressure on the parietal peritoneum, uterine ligaments, urethra, bladder, rectum, lumbosacral plexus, fascia and muscles of the pelvic floor increase the intensity of pain.

Neural pathway of pain
The uterus and cervix are supplied by afferents accompanying sympathetic nerves in the uterine and cervical plexuses, the inferior, middle and superior hypogastric plexuses and the aortic plexus. The small unmyelinated 'C' visceraffibres transmit nociception through lumbar and lower thoracic sympathetic chains to the posterior nerve roots of the 10th, 11th and 12th thoracic and also to 1st lumbar nerves to synapse in the dorsal horn. The chemical mediators involved are bradykinin, leukotrienes, prostaglandins, serotonin, substance P and lactic acid. As the labour progresses severe pain is referred to the dermatomes supplied by T10 and L1.

In the second stage, the direct pressure by the presenting part on the lumbosacral plexus causes neuropathic pain. Stretching of the vagina and perineum results in stimulation of the pudendal nerve via fine, myelinated, rapidly transmitting 'A delta' fibres. From these areas, the impulses pass to dorsal horn cells and finally to the brain via the spino-thalamic tract.

The stress response to pain in labour
Segmental and supra-segmental reflex responses from the pain of labour may affect respiratory, cardiovascular, gastro-intestinal, urinary and neuro-endocrine functions. Respiratory - Pain in labour initiates hyperventilation leading to maternal hypocarbia, respiratory alkalosis and subsequent compensatory metabolic acidosis. The oxygen dissociation curve is shifted to the left and thus reduces tissue oxygen transfer, which is already compromised by the increased oxygen consumption associated with labour.

Cardiovascular - Labour results in a progressive increase in maternal cardiac output, primarily due to an increase in stroke volume, and, to a lesser extent, maternal heart rate. The greatest increase in cardiac output occurs immediately after delivery, from the increased venous return associated with relief of venocaval compression and the autotransfusion resulting from uterine involution.

Hormonal - Stimulation of pain results in the release of beta-endorphine and ACTH from the anterior pituitary. Associated anxiety also initiates further pituitary response. Pain also stimulates the increased release of both adrenaline and noradrenaline from the adrenal medulla which may lead to a progressive rise in peripheral resistance and cardiac output. Excessive, sympathetic activity may result in incoordinate uterine action, prolonged labour and abnormal fetal heart-rate patterns. Activation of the autonomic nervous system also delays gastric emptying and reduces intestinal peristalsis.

Metabolic - Maternal: During labour, glucagon, growth hormone, renin and ADH level increases while insulin and testosterone level decreases. Circulating free fatty acids and lactate also increase with a peak level at the time of delivery. Fetal: Maternal catecholamines secreted as a result of labour pain may cause fetal acidosis due to low placental blood flow.

Severity of labour pain
The severity of labour pain varies greatly among women in labour. If women are asked during or shortly after birth to score their labour pain most rate it as severe while few mention little or no pain. Using the McGill pain questionnaire, Melzack et al in Montreal, Canada, found that labour pain usually rated a high score particularly among primiparae, those with a history of dysmenorrhoea and those belonging to low socio-economic status.

Principles of pain relief
The essentials of obstetric pain relief are:
Simplicity
Safety
Preservation of fetal homeostasis

Women who are given any form of analgesia should be monitored closely. After spinal or epidural anaesthesia they should be monitored with frequent measurements of blood pressure, level of consciousness and maternal oxygen saturation by pulse oximetry.

Role of the Anaesthesiologist

Anaesthesiologists do have a role antenatally. They should be ready to answer the mothers' questions about the methods of analgesia. It is important that women with serious underlying chronic disease should be assessed antenatally by the anaesthesiologist to adopt a management plan before the onset of labour. Good communication between obstetrician, physician, haematologist and any other relevant specialist can help the anaesthesiologist in the management of high-risk pregnancy.

History of pain relief

Ancient methods of pain relief included various plant-derived sedatives, acupuncture and physical methods such as binding. In 1847 James Young Simpson administered the first obstetric general anaesthetic using ether. In 1853 John Snow delivered Queen Victoria's eighth child under chloroform. In 1881 Stanislav Klikovitch described the use of nitrous oxide for labour in Russia. In 1902 morphine and hyoscine was first used in labour. Pethidine was first used in 1940. In 1931 Eugen Bogdan Aburel, Romanian obstetrician, described continuous caudal plus lumboaortic plexus blocks in labour. In 1945 Curtis Mendelson described the syndrome of acid aspiration under general anaesthesia for caesarean section. In 1949 Cleland described continuous lumbar epidural block in labour. In 1958 Ferdinand Lamaze published his book suggesting that pain was a conditioned reflex triggered by uterine contractions, and that psychoprophylaxis could reduce pain. In 1961 Brian Sellick described cricoid pressure as a means of preventing gastric aspiration.

Psychological methods of pain relief

Methods of psychological analgesia can be divided into three broad categories:
- Natural child birth - the Read method.
- Psychoprophylaxis - the Lamaze technique.
- Hypnosis

Each technique claims the elimination of pain without any harm to the mother, the baby or to the progress of labour and without the need for chemical analgesia. All require adequate antenatal preparation. Still most women experience severe labour pain. Furthermore, psychological analgesia can place increased demand on the staff.

Support during labour

A friendly atmosphere in the labour room is preferable to help a woman to cope with pain. Homely surroundings help to allay anxiety and reduce the need for pharmacological analgesia.

Hypnosis. Hypnosis (hypnos, sleep) can produce analgesia and amnesia during labour and delivery for some selected patients. Only about 25% of women however are suitable as deep trance hypnotic subjects. And the technique relies on extensive preparation.

Bio-feedback. This is borderline between psychological and physical methods of analgesia. Relaxation is a major component of psychological preparation for child-birth and is claimed to relieve pain, reduce anxiety and speed labour.

Physical methods of pain relief

Transcutaneous Electrical Nerve Stimulation (TENS). TENS was introduced to relieve pain in childbirth in the early 1980s. Since then the use of TENS in labour has become increasingly popular as it is simple to use and is non-invasive. The mode of action depends on the two principal theories. One that A-fibres are stimulated by the electrical stimulation preventing the transmission of afferent noxious stimulus originating from C-fibres, the other that the electrical stimulus increases endorphines and enkephalins within the system. TENS electrodes are applied over the dermatomes supplied by T10 to L1. The TENS machine then gives a low background stimulus which can be augmented at the time of each contraction. It has been observed in clinical practice that TENS may provide limited pain relief during the first stage of labour. Meta-analysis of randomised controlled trials of TENS in labour does not,
however, confirm its efficacy. Acupuncture. Mentioned in the literature in 581 B.C. and widely practiced in China. Acupuncture is not used for childbirth in China, however, and there are no acupuncture points described for pain relief in labour. Water (bath or shower). A bath or shower is relaxing and should be encouraged. There has been enthusiasm in some quarters to extend this to the delivery of the baby under water and many maternity units have the facility to offer water birth. However, while its use during the first stage of labour is not discouraged, very few units would encourage the use of the birthing pool for the delivery of the baby. At present there is little evidence to support the use of immersion in water during labour.

Inhalational analgesia

Several inhalational agents, both gaseous and volatile, have been used successfully in labour. The earliest to be used were ether, chloroform and cyclopropane, followed by trichloroethylene and methoxyflurane. Enflurane, isoflurane and desflurane are more recent additions. Analgesia during labour can be provided by the inhalational anaesthetic agents in subanaesthetic concentrations thus relieving pain whilst maintaining maternal consciousness and avoiding regurgitation or aspiration of stomach contents. In fact, the competence of the upper oesophageal sphincter is well maintained under light general anaesthesia, although lost under mild sedation with barbiturate or diazepam. Inhalational agents readily cross the placenta and the concentration in foetal blood soon approaches that of the mother but, since these agents are excreted almost entirely through the lungs, they are readily excreted from the newborn.

Inhalational analgesia depends on the analgesic strength of the agent and on how quickly it reaches analgesic concentration after the start of inspiration. A rapid offset with complete elimination between contractions would prevent accumulation completely. Nitrous oxide is the best match in current use. Various portable machines exist for administration of nitrous oxide blended with oxygen through an on-demand valve. Nitrous oxide concentrations can be varied from 0 to 75% in oxygen. For self-administration, a concentration above 50% nitrous oxide should not be allowed. Entonox, which is a mixture of 50% nitrous oxide and 50% oxygen is most commonly used.

Ether has several side effects including potent emetic effects with an unpleasant pungent odour, irritant to the respiratory tract and explosive. Chloroform has a pleasant odour, is non-irritant, more potent and faster acting than ether but has undesirable, dose-related side effects, namely arrhythmias and liver damage. Methoxyflurane and trichloroethylene have been used for analgesia in labour, but have been withdrawn for other, nonobstetric, reasons. Enflurane and isoflurane have been given via a draw-over vaporiser in subanaesthetic concentrations to relieve pain in labour. The usual concentrations, in oxygen, of enflurane and isoflurane for self administration are 0.3-1% and 0.2-0.7%. Such concentrations will not change uterine contractility or responsiveness to oxytocin. The neonate is not affected by these analgesic concentrations of these inhalational agents. Enflurane, however, causes long-term drowsiness so was never popular. Both the agents are expensive and since neither shows a significant advantage over entonox in terms of analgesia they are unlikely to be widely used on their own.

Desflurane is the newest volatile agent to be applied in labour. The chief advantage of this agent is rapid onset and offset of action, however it is expensive and since it has not been shown to provide superior analgesia to entonox, it is unlikely to become a popular agent for labour analgesia.

Systemic opioid analgesia

Opioids have been used for anaesthesia in labour for hundreds of years. However, it was not until the early twentieth century that techniques deliberately employing the analgesic effects of the opioids gained major attention. Unfortunately, dosage and effect are limited by maternal and neonatal side-effects, so that only moderate pain relief could be obtained with these drugs.

Pethidine has become the most commonly used and widely investigated systemic opioid in labour. It is principally a muagonist but of a low potency. Administered as hydrochloride in a dose of 75-100mg intramuscularly it reduces labour pain by about 25%. Delayed gastric emptying is a prominent feature. Respiratory depression is not usually observed in women who receive pethidine, because contractions continue to be painful and to provoke hyperventilation. However hypoxic episodes have been observed probably associated with significant underventilation between contractions. The major metabolite, norpethidine, is itself active, and has convulsant properties. Thus, pethidine may be inadvisable for use in fulminating preeclampsia or eclampsia, particularly in repeated doses. Morphine fell from favour in the first half of the twentieth century, in part because of its association with "twilight sleep" and in part because of its addictive side effects.

Meptazinol is a mixed opioid agonist/antagonist, act primarily at the kappa receptor. It is given in a dose of 100-150mg intramuscularly every 2-4 hours. In high doses it has dysphoric side effects and also produce nausea and
vomiting. The antagonist properties of meptazinol may cause withdrawal in parturients dependent on mu-agonists. It has a reduced potential to cause respiratory depression.

Buprenorphine is a partial agonist acting selectively at mureceptors. It is about 20 times as potent as morphine and has a high affinity for opioid receptors and slow dissociation from them. It has a capacity for self-antagonism, which tend to produce a biphasic time course of action. This may be observed for both analgesia and respiratory depression. It appears to have a long duration of action and though side effects are rare, when nausea and respiratory depression do occur they can be exceedingly persistent and difficult to reverse.

Nalbuphine is a synthetic mixed mu-agonist/antagonist and a kappa-agonist. For analgesia in labour it is given in doses of 10-20mg intramuscularly. Maternal or foetal respiratory depression is less likely with nalbuphine due to the ceiling effect. The chief disadvantages of this drug are sedation and dysphoria. Fentanyl primarily acts on mureceptors and is approximately 80-100 times as potent as morphine. It has a rapid onset action and shorter duration of action. The peak analgesic effect occurs within 5 minutes and the duration of effect is about 30 minutes after 1 mcg/kg administered intravenously. Fentanyl is principally bound to albumin which favours its transplacental transfer. For analgesia in labour 50-100mcg/hour is required, given in increments of 10mcg IV.

Tramadol is a weak mu-agonist that has been prescribed in labour in doses of 50-100mg 4 hourly. The incidence of nausea is more common with tramadol than with pethidine or morphine17. Butorphanol is a synthetic narcotic given as a 1-2 mg dose which lasts 3 to 4 hours. Neonatal respiratory depression is reported to be less than with pethidine18.

**Patient-controlled analgesia**

Patient-controlled analgesia with intravenous administration of opioid analgesics was assessed for obstetric pain as early as 197019. The patient's ability to control the analgesic administration may produce pharmacological as well as psychological benefits.